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ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

THE ENDOCRINES IN OTOLOGY.*

DR. CHARLES E. DE M. SAJOUS, Philadelphia.

In expressing my appreciation of your invitation to address you this evening, I wish to emphasize that it is also a source of deep gratification to find myself among workers in a field closely connected with laryngology which I practiced up to some thirty years ago, and through which my attention was first drawn to the internal secretions.

To interpret with any degree of lucidity the clinical relationship between the few common aural diseases which the limits of a paper will allow me to review and the endocrines, it was found necessary to ascribe to these glands the functions which I attributed to them in 1903 and 1907. Originally based upon a comprehensive analytical study of all data bearing upon these organs contributed by the various medical sciences and personal researches before any deductions were at all formulated, their correctness has been demonstrated in many respects, as will be shown, by the experimental and clinical labors of many other observers.

Unfortunately, when we enter the field to which I have devoted most of my labors,—the connection between the endocrines and diseases other than the very elucidative endocrinopathies proper (hence the title of my work "Internal Secretion and the *Principles of Med-*

*Address delivered by invitation before the Otological Section of the New York Academy of Medicine, March 17, 1921.

icine")—the ever-growing number of theories unsupported by what Huxley once termed "naughty facts" concerning the functions of the endocrines, becomes a source of considerable embarrassment. It is such an experience, in fact, which led Professor Halsted, of Johns Hopkins, to remark after an effort to study an obscure surgical problem: "It must be evident to everyone that there reigns the greatest confusion on the subject of the functions of the glands of internal secretion." It is to avoid for you the bogs into which such a condition of affairs might drive you, and in the hope of rendering your labors fruitful from the start, that I venture at all to refer to this phase of the subject.

Even the branch of medical science which to us clinicians should act as mentor and guide in all that which constitutes function, physiology, seems to dominate in the direction of disharmony. Take, for example, the functions of the adrenals. For many years, we were taught that the purpose of the secretion of these organs was to sustain the tone of the cardiovascular system, and that as a therapeutic agent it would raise the blood-pressure. But other psysiologists found that far from raising the blood-pressure, adrenalin, particularly in small doses, lowered it. We were also told that the adrenal secretion possessed antitoxic properties, but physiological experiments were advanced to show that it did not. Even Cannon's well-known emergency theory in which fright and excitement is considered as a cause of increased activity of the adrenals has been attacked. These and other discrepancies led Swale Vincent, now professor of physiology in the University of London, to conclude as late as 1917 that "we know nothing of the functions of the adrenal body regarded as an organ on its own account." It is obvious that physiology thus interpreted would hardly aid the clinician in elucidating any disease, general or local.

If, however, we treat the question with harmony as our watchword, quite another state of affairs asserts itself. We then find that far from being erroneous, all the functions described, mutually antagonistic though they seem, are entitled to a legitimate place. This, however, is subject to one condition, viz., that they be considereed as but expressions or manifestations of a *fundamental* function submitted by myself long ago.

Some of you may recall that in 1903, I attributed to the adrenals an all-important function: that of endowing the hemoglobin with the power to take up oxygen in the pulmonary air cells and of converting it into oxyhemoglobin. The adrenals thus became the governing organs, as far as oxygenation was concerned, of meta-

bolism throughout the entire body. This conception not only met a need long emphasized by many European physiologists,—that a secretion capable of clutching, so to say, the oxygen from the alveolar air, was a *sine qua non* of the respiratory function—but it also fulfilled every condition of that function. Thus, the adrenal product or adrenin was found to increase the oxygen intake and the carbon dioxide output; to increase both the depth and rate of respiration, also the heat production, temperature, etc. That the adrenals secreted enough adrenalin to meet the needs of so important a function was shown by Stewart and Rogoff,[1] who found that the spontaneous liberation of this substance was 0.0003 to 0.001 gm. per kilo of animal,—thus making 30 to 100 c.c. in the twenty-four hours —ample to meet the needs of the function; Kariya and Tanaka[2] found adrenalin capable of acting as hemoglobin, while Menten, at the University of Chicago, observed that venous blood under proper osmotic conditions could be converted into arterial blood by the addition of adrenalin, the spectroscope showing the typical increase in the intensity of the oxyhemoglobin absorption-bands. Briefly, these and many other data which will be spared you, have shown that the function I had attributed to the adrenals in 1903 was the fundamental one of these organs.

If we now apply this function to the different roles attributed to the adrenals by various physiologists, its harmonizing influence will reveal itself. Thus, large doses of adrenalin, by increasing the oxygenation and metabolism of the cardiovascular muscular elements, cause a general rise of the blood-pressure and may thus maintain vascular tone, as shown by Oliver and Schäfer in 1894; smaller doses, however, through the same process, only cause constriction of the ends of the vessels which are supplied with nonstriated fibers, the terminal arterioles and the heart, as they maintained. This also causes a rise, though passive, of the blood-pressure, since the constricted arterioles dam up, as it were, the blood behind them and increase the tension in the larger vessels. The apparently destructive observation of Moore and Purinton that minute doses cause dilatation of the arteries is also sustained, for such doses affect primarily, owing to their diminutive caliber, the vasa vasorum, thus reducing the arterial blood supplied to the muscular coat of the vessels and causing their relaxation. The antitoxic theory of Abelous and Langlois also finds its normal explanation, for oxygenation and metabolism are necessarily the fundamental processes upon which the defensive mechanism depends for its efficiency, and it is well known that cases of hypoadrenia are very

vulnerable to infections. Cannon's emergency theory also becomes a normal feature of oxygenation and metabolism, for a prompt increase of these functions imposes upon the adrenals the need of the increased secretory activity observed, for active and effcient defense.

On the whole, it is obvious that the discord among physiologists is only due to the fact that although their observations were exact, each describes a different manifestation of a fundamental function, oxygenation, which, from my viewpoint, the adrenals govern.

What is the practical bearing of this function in otology? Limiting ourselves for the present to the systemic phenomena observed, patients are frequently encountered who respond poorly to practically every remedial measure attempted, pharmacological, local or surgical. Such cases, in many instances, suffer from a condition which I have termed "hypoadrenia," *i. e.*, deficiency of adrenal activity. A child of this type exhibits pallor, a pasty skin, flabby musculature, low circulatory and cardiac tone, cold extremities, deficient or capricious appetite and emaciation, unless the thyroid be likewise markedly deficient—for the activity of all endocrines is reduced when either one of them is deficient—when adiposis may coexist. Myasthenia, constipation through atony of the intestinal muscles and occasionally enuresis from a similar condition of the sphincter muscles, are also common. Most of these symptoms are also met in adults and in the aged, their occurrences in the latter being favored by the usual senile atrophy of the adrenals, which greatly reduces their secretory activity. Temporary functional hypoadrenia may result from physical fatigue, as often observed during the European war. It may even cause death, the adrenals being then found depleted of their chromaffin substance.

In all such cases, the deficient oxygenation incident upon the adrenal deficiency not only renders the patient vulnerable to infections, but also makes of him a poor operative risk when meningeal complications are to be feared. Moreover, such patients constitute a large proportion of the chronic cases in which purulent exudates prevail.

Turning to the thyroid, the discord is not so great. Physiologists and clinicians are in accord concerning the influence of this gland upon metabolism. Kendall, to whom we owe thyroxin, by far the most perfect of the thyroid active principles yet produced, states in this connection:[3] "The changes occurring in a patient with thyroid deficiency, or in an experimental animal by the administration of thyroid, are so great that practically every cell within the animal

organism is changed. The effects are felt throughout the nervous system and the circulatory system, the rate of metabolism is enormously increased, and even the skin and hair show revolutionary changes."

How does the thyroid promote this active metabolism? Kendall attributes it theoretically to the deaminization of amino-acids. But this does not meet our observations as clinicians, for it fails to account for the striking increase in the intake of oxygen caused by thyroid gland—75 per cent. in some instances; or for the stimulation of growth forces; or for the increased elimination of phosphates (phosphoric acid in the ash); or for the action on reserve fats which, when exhausted, is followed by breaking down of the nitrogenous tissues themselves. All these effects are explained, however, by the view personally submitted years ago, that the phosphorus of all nucleins, beginning with the phosphorized fats, was the element mobilized by the active principle of the thyroid hormone, iodine. I then urged that the thyroid iodine was contained in the blood-platelets—a view since confirmed by others—and that these platelets were secreted by the red corpuscles along with the adrenal oxidizing principle which I termed "adrenoxidase," a view also sustained by many available data.

The nuclear phosphorus is the fundamental factor of all cellular activities, but it lies relatively dormant, though potential, in the nuclei or nucleoli of the cells until mobilized, or better, sensitized by the thyroiodine. Oxygen being supplied simultaneously, the normal vital activity of the cell is dependent upon the perfect equipoise of the thyroid and adrenal hormone present. If there is deficiency of the latter, hypoadrenia occurs, we have seen, but if it is the thyroid principle which is deficient, we have cretinism in the child or myxedema in the adult. In such cases the nuclear phosphorus is sensitized inadequately and oxygenation being correspondingly reduced, as well shown by the low oxygen intake and carbon dioxide output, cellular metabolism is slowed, to such a degree in some cretins that they have been termed "human plants." You all know the marvelous effects thyroid gland sometimes produced in such patients.

Otologists and laryngologists are seldom called upon to treat such cases. Those they do encounter frequently, however, belong to the larval group, so well described by Hertoghe, but which may occur in many gradations of severity. These patients belong to what might be called the torpid and morbid class—languid, constantly fatigued, somnolent on rising—all of which symptoms tend to disappear as

the day wears on. They sometimes complain of headache and backache, usually between the shoulder blades or of sacrolumbar pains which rest in bed tends more to aggravate than to improve. Their temperature is low, and their hands are flabby, cold and sometimes damp, as are apt to be the feet, the latter being occasionally of the type termed "flat-foot," and requiring central support. The skin is sallow, pale and waxy, causing the patient to appear older than his or her age, and suggesting an underlying renal trouble. Alopecia, beginning at the forehead and tending towards the occiput, is common in adults, the hair in marked cases being coarse, dry and brittle. The eyebrows tend to become shortened, at the expense of the outer ends. Marked cases may also show cutaneous infiltration as in myxedema, perhaps an increase of weight and even pads of fat over the clavicles. The teeth tend to become carious and loose and are particularly prone to become tartrous, with pyorrhea alveolaris as an ubiquitous result.

Such cases are particularly liable to nasopharyngeal infections with aural complications through the Eustachian tubes. This is due to the lowered germicidal activity of the vault where the defensive barriers are of the utmost importance owing to the constant inhalation of bacteria-laden air. Why this vulnerability to infection, so important to the otologist?

I must confess that I regard as exceedingly unfortunate, in view of the incalculable harm it does, the neglect in this country of the role of the thyroparathyroid apparatus in the immune chemistry of the body, now fully recognized particularly on the continent of Europe. It is not only a function which may be utilized to treat successfully many obscure disorders, but one which when abnormally active, as in hyperthyroidism and dementia precox, may cause great suffering, insanity and death.

Although Horsley, in 1892, showed that the role of the thyroid in catabolism included the breaking down of toxic waste products, of metabolism, the theory that the thyroparathyroid mechanism took part in our defensive functions against infections and exogenous pathogenic agents was introduced by myself in 1903 and further developed in 1907. I then urged that it played the role of the sensitizing agent described originally by Denis and Leclef, and which Wright and Douglas termed "opsonin." This has been confirmed at the Pasteur Institute, the Bacteriological Institute of Liege, also in Russia and Italy. Leopold-Levi and Rothschild,[4] of Paris, for instance, state in this connection: "Sajous has attributed among the functions of the thyroid body a role which he assimilates to the

opsonins and to antitoxines. More recently Fassin, Stepanoff and Marbe have confirmed on their side, the influence of the thyroid on the blood's asset in alexins and opsonins." McCarrison, of India,[5] to whose labors we owe much in this connection, states in a review of the question, beginning with my labors: "The fact that the antitoxic and bactericidal resources of the body are largely dependent on the functional perfection of the thyroparathyroid mechanism is, I believe, as clearly established as is the influence of this mechanism on metabolism." His excellent book upon the Thyroid Gland fully confirms this conclusion.

Many bacteriological studies have shown that the thyroid hormone seems to sensitize every pathogenic organism, precisely as it does all tissue cells, to the bactericidal action of antibodies, while an equally large number of investigators found that hyperplasia of the gland was present in febrile diseases though absent in the nonfebrile. Farrant,[6] after studying the subject in a large number of diseases, including tuberculosis and syphilis, states for instance, that "repeated injections of diphtheria toxin lead not only to the production of thyroid hyperplasia, but also to the formation of antitoxin," thus confirming my earlier contention.

Given then a case in which hypothyroidism exists along with some deep-seated suppurative aural disorder which cannot be eradicated by local treatment, the desired result will fail to be attained unless thyroid or iodine in some form is administered internally. Irrespective of an increase of antibodies evoked by other means, the pathogenic organisms will remain masters of the situation unless they are prepared for their proteolytic action by the sensitization which an adequate amount of thyroid hormone insures. As recently shown by Koopman[7] in animals, thyroid therapy, moreover, actually increases the antibodies themselves. Its influence on immune chemistry is thus double—as sensitizer and indirect antigen.

Thyroid gland possesses another property, particularly if given with suprarenal gland or hypophysis sicca, that of increasing the coagulability of the blood. Even in cases in which hemophilia was present I have seen the coagulation time reduced to three minutes, thus insuring a dry operation where otherwise any attempt in this direction would have proven disastrous if at all attempted.

Another function of interest to otologists which the thyroid body carries on, through its parathyroids as shown by Erdheim, is that of promoting calcium metabolism. This is well shown by the action of total thyroid gland in cretinism, in which growth is so rapid at

times that the bones become soft and curved unless calcium is supplied simultaneously. Again, if the parathyroids are removed in animals, ossification is decreased to such a degree that the teeth become soft and break down, while broken bones heal very poorly if at all. This function is also well shown in osteomyelitis, where the use of thyroid gland often causes marked improvement. We will presently see the bearing of this function.

A brief allusion is necessary in the present connection to the pancreatic internal secretion as a factor in the defensive process. Besides its role in carbohydrate metabolism, the pancreas supplies enzymes which take part in protein metabolism and in our defensive functions. Trypsin, in fact, is the active agent which both in the phagocytes and in the blood digest bacteria precisely as it does proteins. Opie[8] writes, for instance: "The ability of the blood to remove injurious material is dependent on the possession of proteolytic enzymes. Peculiar to the polymorphonuclear leucocytes is an enzyme which like trypsin, exerts its digestive action in an alkaline medium." Abderhalden[9] also states: "Each separate cell, with very few exceptions, disposes of the same or similar ferments as those secreted by the digestive glands in the intestinal canal." In keeping with my own views expressed several years earlier, he also termed the digestive enzyme a "defensive ferment," thus "bringing," he held, "the so-called reactions of immunity into close line with processes that are normal and consequently familiar to the cells."

What is the connection between trypsin thus interpreted and the adrenal and thyroid hormones previously reviewed?

Trypsin at the normal temperature merely carries on the catabolic phase of metabolism. When, however, pathogenic substances, bacteria, toxins, etc., appear in the blood or tissues, the thyroid and adrenals become overactive, thus raising the temperature. What happens? As every chemist knows, the activity of ferments is increased by heat. In the case of trypsin, its activity may be raised to 104° or 105° F. In the presence of pathogenic substances, toxins, bacteria, etc., the rise of temperature produced by the excess of thyroid and adrenal hormone increases the digestive activity of the trypsin which, in both the phagocytes and the blood, digest the harmful agent. You have, of course, recognized in this process a kinship to fever. It is my firm conviction that this phenomenon, now admittedly obscure, will prove to be a defensive reaction carried on mainly by certain ductless glands. The importance of this process will be duly emphasized presently.

Upon the pituitary I shall have but little to say because it would take us too far afield. As some of you may know, I am not a believer in the pituitary secretory theory. Before long I shall reaffirm my views in this respect and show that time has only served to strengthen my position. As you are aware, the hypophysis sicca, the preparation which is supposed to represent the active secretory principle of the posterior lobe, is the only product recognized in the U. S. Pharmacopeia. Yet, in accord with my previously expressed view, Biedl and also Swale Vincent do not recognize in this lobe any of the histological characteristics of a gland. Again, the colloid formed in it was thought to be a secretory product. But Kendall found in his work upon the thyroid that colloid was not a secretion at all. This does not in the least militate against the pharmacological value of posterior pituitary preparations. In fact, it explains their practical usefulness by revealing their true identity for, as much clinical and bio-chemical evidence tends to indicate, they contain the constituents of both the medulla and cortex of the adrenals and are therefore far more efficient than adrenalin to counteract circulatory failure, and also more lasting in their effects. It is in this connection that their action will be referred to when the therapeutics of the various aural disorders will be reviewed.

As regards the other ductless glands, the thymus, pineal, etc., our knowledge is still too uncertain to permit anything but conjectures in the present connection. They will not, therefore, be considered.

I will now turn to the practical application of these functions, limiting my remarks, however, to the general principles involved in the more common disorders of the aural apparatus. A feature worth emphasizing in this connection, is that when the functions of the various endocrines are interpreted as above, nothing actually established on a sound basis concerning general or local diseases has to be sacrificed, while much appears to be added to our knowledge of the pathogenic and therapeutic principles involved.

Recalling the defensive role of trypsin, deafness resulting from the acute suppurative otitis media following acute infections of childhood, scarlatina, diphtheria, etc., is due not to the direct action of the pathogenic bacteria themselves, but to the local autolysis or digestion of the drum and other structures by the trypsic ferment which should serve to destroy solely the local bacteria and their toxins. Indeed, bacteria are not necessary to cause such lesions. This is shown by the fact that similar lesions of the cardiac valves and endocardium may be produced by uranium nitrate, potassium bichromate, the arsenates, spartein and other drugs given *internally*

in sufficient doses, as shown by Christian and Walker.[10] All these agents are powerful excitors of a general defensive reaction. This applies also to the diseases of children in which the nasopharynx is the primary seat of the morbid process, the infective bacteria entering the tympanic cavity through the Eustachian tube. Here also the aural disorder usually develops at the height of the nasopharyngeal process, when the fever is highest. This fact and practical experience suggest that the severity of the lesions might be mitigated, and the resulting deafness perhaps prevented by employing, when the fever is high, and in addition to the customary treatment, a measure which serves to prevent local lesions in endocarditis, pleuritis, etc., *i. e.*, hypodermoclysis. The abnormal viscosity of the blood which is a prominent factor in the production of the local lesions, is counteracted; its osmotic properties are improved, thus facilitating a reduction of the vascular tension by an induced overflow of plasma into the lymphatics. Freer secretory activity of all mucous membranes is induced, including that of the Eustachian tube and tympanic cavity, thus protecting it and staying the autolytic process.

Should we employ organic preparations during the acute febrile period of these diseases? A negative answer suggests itself since they would simply increase the endocrine agents causing the proteolytic process, and thus activate the autolysis to which the lesions are due.

The case is different, however, when the febrile period has passed and the adrenals and thyroid in particular are exhausted after the excessive stimulation to which these organs have been subjected during the febrile period. When death occurs at this time the adrenals are found extremely poor in medullary substance—so poor in some instances as to respond negatively to the chromaffine test. Conversely, when adrenal failure threatens, as shown by weak cardiac impulse, hypothermia and increasing weakness, the slow intramuscular injection of 8 minims of the 1-1000 solution of adrenalin in a syringeful of saline solution, repeated two or three times daily, restores defensive conditions within safe limits. When injections cannot be used, the insertion under the tongue of a triturate pellet containing 1/60 grain of adrenalin hydrochloride repeated as needed, will serve a good purpose. Thyroid should be avoided as long as the heart is weak, owing to its action on the depressor nerve which tends to lower the vascular tension. When the blood-pressure is low the hypophysis sicca in 1/10 grain doses is better than even adrenalin. Many clinicians, in fact, have found it advantageous

to combine these agents, injecting four drops each of adrenalin and pituitrin. By thus raising the vascular tension, the aural apparatus receives its normal supply of arterial blood, the bearer of the agents, both cellular and humoral, which carry on the process of repair.

In the acute suppurative otitis media due to causes other than acute febrile infections—those commonly attributed to "cold," naso-pharyngeal disease, including chronic tonsillitis, carious teeth, etc., and in which no perforation of the drum usually occurs, unless it be temporary for the evacuation of pus, conditions are different in the sense that the febrile process, the temperature hardly exceeding 103° F., is not sufficiently active to produce autolysis leading to tissue necrosis. The tympanic mucosa is simply more or less inflamed, with a correspondingly marked exudation of muco-pus or pus, all kept up by the presence of the pathogenic bacteria. When any degree of adrenal or thyroid insufficiency exists, the defensive reaction is inadequate and the suppurative process persists notwithstanding the local measures adopted.

Three decades ago we administered calomel to clear the intestinal canal and produce what was then called its "alterative" effects. Used besides familiar local measures, we obtained good results, particularly if the calomel was continued in what was then termed "tonic" doses, about 1/12 grain three times daily. A comprehensive research upon the action of mercurials revealed what seemed to me at least, the explanation of the excellent effects produced, particularly in the treatment of diphtheria, in pre-antitoxin days; it showed that mercury was perhaps the most powerful stimulant of the ductless glands at our disposal. Calomel is also, owing to this fact, a potent adjuvant when antitoxin fails to act. Thus, on a trip from Naples, the ship surgeon imparted to me the distressing fact that he was burying at sea two or three children every night, owing to an epidemic of diphtheria, notwithstanding the free use of standard antitoxin. The immigrants were of a poorly developed type from Hungary, unable to react against the infection and thus assist the antitoxin. Calomel given by the mouth, however, in addition to the antitoxin, at once stopped the deaths. Hence my predilection for the internal use of calomel to this day.

Another measure employed at the time was the application of heat, hot compresses or the hot water bag over the affected ear, with a wad of cotton dipped in tincture of belladonna, lit with a match to warm it, or a couple of drops of a 4 per cent. solution of cocaine in the external meatus to quiet the pain. But the external heat, faithfully applied, seemed always to hasten the cure. Why? This

is usually explained by the statement that blood is drawn to the surface; but this hardly accounts for the curative influence. The latter becomes clear, however, when we recall that heat increases the bactericidal activity of the defensive enzymes; and that the external heat supplied increases that furnished by the interaction of the endocrines and the nucleins. In other words, we are enhancing the local fever and hastening the destruction of the local pathogenic bacteria and their toxins.

In both acute and chronic otitis media, thyroid and hypophysis sicca or suprarenal gland are indicated, particularly where the curative process is indolent, and as powerful aids to the local measures adopted. This applies as well where the mastoid process is involved. Both here and where the ossicles are the seat of caries or necrosis, calcium, the metabolism of which in part depends upon the functional efficiency of the thyroid and thymus, using calcium lactate in 5-grain doses, should prove helpful. Thymus gland in 5-grain doses seems also beneficial in debilitated children in whom the gland itself is inadequately developed or is undergoing too rapid involution.

Nerve deafness seems likewise to be elucidated by the functions of the endocrins. You are all aware of the fact that the entire sympathetic system, including its ganglia, contains chromaffine substance, that is to say, adrenal medullary substance. In 1903, methylene blue and other injections led me to conclude that the adrenal principle circulated in the axis cylinder of all nerves, gravitating therein as does tetanotoxin, in order to carry on oxygenation. This has been sustained by the labors of others, both as to the presence of the adrenal principle and as to the occurrence of active oxygenation in nervous tissue. As previously stated, the thyroid principle, its organic iodine, accompanies the adrenal principle. The axis cylinder thus becomes a minute canal or aggregate of canalicular fibrils, which transmit the oxidizing and sensitizing substances to the phosphorus-laden nucleus and nucleolus of the cell-body of the neuron and the myelin, also composed of phosphorized fat. The functional activity of nerves, therefore, is carried on precisely as it is in the other tissues of the body.

A few examples will now suggest how nerve deafness might be produced. Thus quinin and sodium salicylate are both constrictors of the peripheral arterioles and thus reduce the volume of arterial blood circulating in the nerves of the acoustic apparatus; again, an excess of tobacco, alcohol and lead poisoning are depressors of the adrenal functions, thus depriving the blood of the oxidizing principle which the acoustic nerves require for full efficiency. A third

cause, old age, produces increasing dullness because, as is well established, the adrenals—as well as the thyroid—undergo atrophy as age adyances. The same explanation applies to the deafness sometimes following acute infections, even though it has not been the seat of destructive lesions, for we now know, that both the thyroid and the adrenals often become the seat of hemorrhages and other changes during the acute febrile process, which greatly impair their functional efficiency.

Briefly, in so far as the ductless glands are concerned, nerve-deafness probably occurs when the acoustic apparatus is either rendered ischemic or deprived of the endocrine substances which sustain their oxygenation and metabolism.

As regards treatment, it is obvious that a clear interpretation of the cause is necessary. It would not do, for instance, to give pituitary or adrenal preparations in deafness due to quinin or the salicylates, for we would further constrict the arterioles and increase the ischemia of the acoustic nerve ends. Here the aim is to increase their blood-supply, which may be done by giving the nitrites, sweet spirit of niter to begin with, or arsenic, which agents dilate the arterioles. In the forms directly traceable to adrenal insufficiency, however, the adrenal principle is the logical agent to use. The hypophysis sicca—which we have seen embodies the action of both the suprarenal cortex and medulla—1/10 grain, with strychnin 1/50 grain, given in a capsule three times daily after meals, are indicated and may prove useful if destructive lesions, or atrophy of the cochlear nerve is not complete. Where there is any degree of hypothyroidism discernible, thyroid gland may be added, 1 grain to each dose. Calcium, the assimilation of which is favored by thyroid, and which has been found by MacCallum to increase the excitability of nerve and ganglion cells, might also be used, preference being given to calcium lactate in 5-grain doses after meals.

Might otosclerosis not be due to some such cause? Gray[11] attributes it to failure of the local blood supply, the result of a condition which he assimilates to anemia. This, in turn, leads to absorption of the bony capsule surrounding the labyrinth and its replacement by spongy bone and impairment of hearing. Suggestive in the present connection is the fact that, using Gray's own words, "in all, at least, in almost all cases, no matter whether the deafness has progressed rapidly or not, or has remained stationary, there comes a time, usually between the ages of fifty and sixty, when the hearing begins to diminish rapidly." He noted this rapid increase in individuals who had no signs of arteriosclerosis. Have we not

here, particularly in predisposed individuals—heredity being frequently traced—the expression of a senile hypoadrenia or of the hypoadrenia which may occur at any age with the resulting impairment of the hemoglobin molecule? Gray found iron beneficial in these cases; but suppose we round out the patient's hemoglobin by giving also suprarenal gland, or hypophysis sicca, or better, injections of pituitrin, which supplies that constituent which enables it to take up more actively the pulmonary oxygen? Might we not then enhance the curative process by increasing the vitality of the aural nervous and vascular mechanism?

In endocrinological work we occasionally see disturbances of the aural apparatus which are quite perplexing, at least to the non-specialist in otology like myself. Thus, cases of clearly defined hypothyroidism sometimes complain of a jumble of symptoms which appear etiologically antagonistic: tinnitus, hallucinations of hearing, rumbling noises, etc. Yet, they all appear to be due to defective intraneural metabolism and vascular relaxation, possibly with the passive congestion incident thereto as additional factor. Others, more advanced, show distinct dullness of hearing. The symptoms all seem to disappear, however, when thyroid treatment is resorted to, particularly if suprarenal gland and hypophysis sicca are given simultaneously. These agents seem undoubtedly concerned with the perpetuation of the life process itself in all tissues, replacing those constituents of the living cell upon which it depends for the utilization of the exogenous elements which the vitamines probably represent. Administered concomitantly, they all seem to contribute a share to the striking improvement noted where endocrine medication is clearly indicated.

REFERENCES.

1. Stewart and Rogoff: Jour. Pharm. and Exper. Therap., 1916, viii, 479-524.

2. Kariya and Tanaka: Jour. Tokyo Med. Assoc., 1913, xxvi, No. 20; Sei-I-Kwai Med. Jour., 1913, xxxii, 10.

3. Kendall. E. C.: Journal-Lancet, 1917, xxxvii, 768.

4. Leopold-Levi and Rothschild: Physiopath. du Corps Thyroide, Paris, 1911.

5. McCarrison, R.: Lancet, 1913, i, 365-373.

6. Farrant, R.: Brit. Med. Jour., 1913, ii, 1363-1367.

7. Koopman, J.: Endocrinology, 1919, iii, 318-320.

8. Opie, E. L.: Arch. Internal Med., 1910, v, 541-568.

9. Abderhalden, E.: Defensive Ferments of Animal Organism, N. Y., 1914, 3rd Ed.

10. Christian and Walker: Trans. Amer. Soc. for Advancement of Clin. Investig.; Boston Med. and Surg. Jour., 1910, clxii, 901-902.

11. Gray, A. A.: Otosclerosis, London, 1917, 1-197.

THE MODERN CONCEPTION OF DEAFNESS AND ITS TREATMENT.*

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Although we have advanced little in our conception of the pathological conditions underlying deafness, our common sense has advanced considerably so that we are less inclined to treat cases along empirical lines. It was not so long ago that all deafened persons were immediately treated to a "blow-out," as soon as the otologist could conveniently arrange his morning appointment. It was either Politzerization or catheterization, with advice to the patient to inflate his own ears, if he could, by puffing out his cheeks and swallowing—the old Valsalva method. Sometimes the otologist connected the patient's ear to his own, with a sounding tube, to see if any air really passed through the Eustachian tube, but often he purposely neglected this procedure because, as far as the patient was concerned, it made little difference. The cases generally fell into two classes, those who actually were improved (and this was a small number) and those that thought they were improved. Whether our newer and more precise methods of treatment will bring about better results, remains to be seen.

Attempts have been made to classify progressive deafness on a pathological basis but without great success. (You will notice that I use the term progressive deafness instead of the time-worn phrase, catarrhal deafness. I also prefer to use the term deafened instead of deaf, to differentiate the congenitally deaf from those who have gradually lost their hearing. This nomenclature is coming into use more and more.) There are various reasons why it is difficult to classify forms of deafness on a pathological basis, the chief of which is that whatever pathological lesions are present during life, change greatly after death. Outside of syphilis, tuberculosis, various forms of suppurative diseases of the middle ear causing deafness and distinct labyrinthine diseases of whose pathology we know, it is impossible to make exact statements of pathological conditions, except that at times we are able to observe adhesions, binding the ossicles together, or between the ossicles and the internal wall, or various aberrant conditions of the drum such as rigidity, relaxation, etc.

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Too much time has been spent in attempting to get at the pathology of the deafened ear; too little time has been spent in studying the pathology of the causative factors and their effect upon the deafened ear. I will admit that the conscientious otologists make a thorough examination of the nose and throat, but unfortunately, few of them avail themselves of the newer instruments of precision, such as the naso-pharyngoscope, and search patiently for the one factor which may be the cause of the condition.

I do not want to seem in too critical a frame of mind but it has been difficult for me to understand why the problem of the deaf has not appealed more strongly to the men engaged in our specialty. Surely no one will gainsay the fact that these unfortunates are worthy of more consideration. From my conversations with many men and a large correspondence with others, I feel that most of them think that it isn't worth while. They take the stand that we never have been able to do anything for the deafened and we never shall. The physician is in the unfortunate position of having his patient feel that he is experimenting upon him without any hope in his own mind that he will be able to do anything worth while. It is because of this haphazard way of treating these cases, that a man named Ferrall wrote an article in the *Volta Review* entitled, "Wild Aurists I Have Met." It was a mean article but only too true from the patient's point of view. In this article Ferrall relates his experiences of going from one ear specialist to another, each one of whom tampered with his ears until his hearing became hopelessly bad. Each one suggested a different operation upon his nose and throat. Each one inflated his ears and after a few treatments, advised him that it wasn't worth while going further. Is it not only human that such a patient should think he has been treated to a course of humbuggery? I answered Ferrall in an article entitled, "Wild Patients I Have Met," the concluding paragraphs from which I quote: "How easily we doctors make our money! All we have to do is to look at a patient, reel off a lot of un-understandable stuff, and collect as much as the traffic will bear. So says the wild patient who has parted with his money after being told things he no doubt does not wish to hear. I wish he could see us day after day, working on dozens and dozens of the poor who can't come to our office—patients who look at us with an awe and respect which is worth more to us than money. I wish he could see us poring over our books, trying to find

some method which is better than the old one, to help the deaf. I wish he could see us in the wee small hours of the morning, wide awake, trying in the quiet around us to devise some new method, some new instrument, that might help. I wish he could hear the groans when the right thought won't come. I wish he could feel the discouragement which every one of us wild aurists feels at times when he wonders whether it is all worth while and when he feels like telling his patient that he had better save the money!"

It is very unfortunate that the interest in the treatment of these cases is lacking and still more unfortunate that certain forms of treatment, which in well-differentiated cases, do a great deal of good, are not being tried. About six years ago I read a paper before the New York Academy of Medicine on relaxed ear drums as a cause of deafness and their treatment by the direct application of cantharides collodion, as advocated by Heath of London. Because Heath had been condemned by our specialists for methods which did not seem quite ethical, no one would use the treatment. This was surely a narrowminded policy which ought to be discarded. The other night I met Dr. Swain, of New Haven, who asked me whether I still used this treatment. I not only informed him that I did, in selected cases, but that I thought more of it than ever. Yet how many otologists are differentiating the relaxed ear drum from the adherent one and treating such cases along the lines advocated by Heath? Perhaps I am a little too severe in my criticism of a class of men for whom I have the greatest respect and admiration, but I am anxious to drive home the point that the differentiation of cases along clinical lines and the proper treatment of cases after this differentiation, has been most sadly neglected.

The clinical diagnosis at the present time is the only satisfactory one and that is open to a great many misinterpretations. But we may amplify this clinical diagnosis by interpreting various self-evident pathological data, such as the abnormal conditions of the nose and throat, the moveability of the drum and ossicles and their physical characteristics, the patency of the Eustachian tube, etc. Such data may be added to, by making the various tests for hearing.

Aside from those lesions which cause complete deafness, there are a number of intermediate stages. On the one hand are those patients who are mildly hard of hearing. On the other hand are those patients who have progressed to the stage where they are very hard of hearing but not totally deaf. The deafness of all

these patients is either due to loss of sound conduction or loss of sound perception. Where the sound conduction is at fault, the lesion is either in the auditory canal or in the middle ear; where the sound perception is at fault, the lesion is in the internal ear, in the auditory nerve or in the auditory center in the brain. Now where is the dividing point between these cases? I admit I do not know, but I do say this—that many cases of so-called perceptive deafness as diagnosed by the usual tests, have been found to be deaf because of a loss of tone of the membrana tympani and the same statement holds true for many cases diagnosed as oto-sclerosis.

In the past, the differential diagnosis of progressive deafness has been determined by various tests with tuning forks, whispered voice, spoken voice, watch, Galton whistle, noise apparatus, etc. Unfortunately in all these tests the personal equation is such a great factor that it is a question how much they are worth. We try the Rinné test, the Weber test, the Schwabach tests and so on and then we test the hearing with various tuning forks, from the low 24 to the high 2048, and finally we employ the Galton whistle to determine the perception of the highest notes. Aside from everything else, the assumption usually is that deafness to the low notes means some interference with sound conduction, while deafness to the high notes means some trouble with the perceptive apparatus. This is far from the case. It is unnecessary to go into an extensive discussion of this matter now. I merely bring out these facts to show you how fallacious one's conclusions may be. A case in point will illustrate this:

A young man of twenty-five came to consult me, stating that he had been almost completely deaf in the right ear for three months. Moreover, on arising in the morning, he became so dizzy and nauseated that it took him some time to right himself. This dizziness, at times, lasted all day. He had a slight tinnitus. Examination showed that the nose and throat were normal except for an edematous condition of the mucosa of the Eustachian tube. The ear drum looked normal. There was no retraction and diagnostic vibration with an electric pump showed that the drum excursions were normal. Hearing tests showed the left ear normal. Voice, whisper and watch could not be heard in the affected ear. Rinné test negative. Weber test lateralized to right side. Schwabach positive. High tuning forks could not be heard. These tests naturally suggested a perceptive deafness, particularly as turning tests showed a hypersensitive labyrinth. I was not sure that all his dizzi-

ness was due to his labyrinth and insisted that he have his eyes examined. Ordinarily a bad prognosis would have been given. I told the patient I was not positive I could help him and that a Wassermann test was most necessary. But before doing anything else, I told him I would dilate up his Eustachian tube and see whether this would relieve any intra-labyrinthine pressure. The tube was gently dilated with a Yankauer applicator, followed by a suitable sized bougie, which was allowed to remain in place one half hour. It was then removed and the ear gently inflated. The sounding tube indicated that the excursions of the drum were within normal limits. His dizziness immediately disappeared and the hearing tests showed a remarkable change. In fact, the tests showed a practically normal ear. I was as much surprised as the patient and told him not to count too much on this one treatment. I asked him to return as soon as he had any further trouble. That is nearly a year ago and he has been perfectly well ever since.

Now what can be deduced from a case like this? One is forced to the conclusion that any change in pressure within the middle ear may cause a change in pressure in the internal ear resulting in a loss of sound conduction and sound perception. Acute cases of this kind are not rare. One encounters them often among individuals who have started an acute inflammatory process of a low grade, after bathing, for example. In such cases, as soon as the normal ventilation of the middle ear is established, the symptoms entirely disappear. But when a patient comes to you, complaining of such symptoms over any length of time, one is inclined to look at the case seriously. I will not go so far as to say that hearing tests should be abandoned, for comparatively speaking, they are of a great deal of value, particularly when they are made at regular intervals on the same individual, by one person. But nine times out of ten, if the same tests are repeated by another otologist, the results will differ.

This brings up another important point. In the severe cases of deafness, one makes his hearing tests and, unless he is very much interested in the subject, invariably gives a bad prognosis. If he has the mental welfare of his patient at heart, he will take the trouble to work out a plan of mental reconstruction in which will be included a certain amount of medical treatment which in itself does little good except to psychologically stimulate the patient. Such treatment is not humbuggery if the physician honestly tells his patient that what he is trying to do is to keep his nose, throat and ears in as good shape as possible, so that he may at least retain the slight amount of hearing he has. Such a patient won't go with-

out any treatment. He is going to find someone somewhere who will do something to his ears, and the honest otologist feels, aside from any sinister consideration, that the patient's mental and physical control is better in his hands than in another's.

But the most important patients with whom we have to deal, from the curative standpoint, are the near deaf, patients that hear a watch on contact with the ear, who hear a whisper at three feet and the voice at a fair distance. They are the ones in whom the process may be arrested or improved. It is precisely this class who are made worse by inflation of the ears with no attempt to eliminate the underlying etiological factors. Here is a case in point:

A doctor, 28 years old, living in Havana, Cuba, came in to consult me a short time ago. He had had a slight tinnitus in the right ear for the past two years with diminution in hearing. The ear felt stuffy. About six months ago he had had a submucous resection performed. The ear symptoms still continued. A prominent otologist of Havana dilated his Eustachian tube for some weeks but there was no abatement of the symptoms. Examination of the nose showed that the submucous resection had been excellently performed. The sinuses were normal. The tonsils were flat, hypertrophied and diseased. Examination with the naso-pharyngoscope showed enormous masses of polypoid tissue hanging from the posterior tips of the inferior turbinates, particularly marked on the right side. The Eustachian tube was markedly congested. Closer questioning revealed the fact that the symptoms were intermittent. He stated that at times his ear would clear up. The Eustachian bougie, after shrinking the tube with cocain and adrenalin, could readily be passed into the middle ear. Therefore, in this case, the inference is clear that the polypoid tips of the turbinates are entirely at fault, and that local treatment of this part, either by fulgeration of the tips or operation, will cure him.

How often we hear patients say that their ears open and close, that when they get into a dryer atmosphere their hearing is better. If one analyzes this statement, he can assure himself that the basic fact is that the reason the patient hears better, at certain times, is because his Eustachian tubes act more normally, thus giving proper ventilation to the ears. But the reason why the tube does its duty at such times, is because the turgescent mucous membranes of the naso-pharynx have diminished in size. Whenever a patient tells me that his hearing acuity is intermittent, I am hopeful that an exhaustive search of the naso-pharynx will reveal a trouble which can be eliminated so that the hearing will remain at the maximum.

Last winter I had a patient with similar symptoms to these related

in the above case. She graphically told me that sometimes she felt something like a marble rolling around in the back of her throat until it fitted into a socket, when she would become stone deaf. A polypoid posterior tip was discovered and removed. The symptoms entirely disappeared. Before operation, I had tried the usual dilatation and inflation, without success. I reported this case before the Medical Society of the State of New York, in March, 1920, in a paper entitled, "Hypertension and Hypotension of the Ear Drum, Associated with Deafness and Tinnitus."

It is unnecessary to say that a thorough routine examination of the nose, throat and ear is desirable in all cases and, from my point of view, more is to be attained by such painstaking examination than by spending time on intricate hearing tests.

In the paper just cited, I went into a thorough explanation of causative conditions which are frequently overlooked. Among these, I mentioned adhesions in the fossa of Rosenmuller which have a tendency to interfere with the normal action of the tubal muscles, polypoid posterior tips of the inferior turbinates which frequently overhang the Eustachian orifice and diseased teeth or buried molars which are a source of continuous irritation to the naso-pharynx.

After all, comparative tests of hearing with all the instruments at our command, mean little to the patient unless he himself feels that his hearing is improved. If the patient feels better, if he hears better or if he thinks that he hears better because of what we have done for him, we have accomplished something, for he goes out into the world in a reconstructed frame of mind and, from a personal, social and economic point of view, he is a different and far more agreeable individual. From my intimate contact with all cases of the deafened I feel that our most important duty is to approach the patient from his point of view, not from our point of view.

Our interpretation of the patient's hearing acuity, by means of the diagnostic aids at our command, by sounding his psychological state of mind, by examination of the middle and internal ear and by analyzing the causative factors which have caused the condition, is most important. But after the diagnosis has once been made, one must not be too discouraging in his prognosis, because there are times when our conception of the seriousness of the condition is faulty and when painstaking treatment over the course of a month or so, will demonstrate that the patient's condition can be greatly improved. I have seen cases where remarkable improvement has taken place in which, in the beginning I felt little or nothing could be done. Let me cite the case of a young woman who came to me nearly ten years ago, with a severe tinnitus in both ears

and hearing so poor that she could not hear a watch on contact with either ear. Former otologists had diagnosed the case as one of oto-sclerosis. I shall not go into a detailed description of the hearing tests. Examination showed a markedly relaxed drum with a stenosis of both Eustachian tubes. She was given what has since then been called the Hays-Heath treatment, which consists of daily applications of cantharides collodion to the drums with dilatation of the tubes, but no inflation. At the end of ten days, the tinnitus had disappeared, and at the end of a month she could hear a watch at ten inches. I have seen her at irregular intervals since that time and am happy to state that the tinnitus has not recurred and that her hearing is still good. Her ear drums were properly tightened but the tubes still have a tendency to stenose.

The citation of this case brings the subject of oto-sclerosis to my mind. Whenever I have the opportunity to discuss this subject with otologists, I always ask "What is oto-sclerosis?" As a pathological entity we know that such a condition exists, but are we able to diagnose it clinically? I am frank to confess that I am suspicious of an oto-sclerosis in certain cases, but I am yet to find the first case in which I can definitely state that there is an oto-sclerosis. Pathologically oto-sclerosis is a rarefaction of bone on the promontory and around the oval window with definite changes in the cochlea. Phillips defines oto-sclerosis as a spongification of the bone of the labyrinthine capsule. He states that the process begins as a change from the normal consistency of the bone to that of compact bone. This is later replaced rather irregularly by the spongy deposits. The spongification takes place particularly in the labyrinthine capsule and around the oval window, eventuating in an involvement of the auricular ligament, and finally in an ankylosis of the foot-plate of the stapes. It is often hereditary. The clinical signs are a rapidly progressive deafness, often in early life. Examination reveals a practically normal drum with a defined pinkish area in the region of the short process. The Rinné test is not definite. Bone conduction is markedly prolonged. Beck, of Chicago, states that he can show definite bone changes on the X-ray plates. Now, if clinical findings could be definitely corroborated by pathological findings, we would have a clinico-pathological complex which would be of value, but the truth of the matter is this: Many cases of deafness are termed oto-sclerosis because they seem to be cases which are getting progressively worse and where treatment is of little use. Inflation has been tried; the nasal and throat abnormalities have been attended to and yet the patient is no better.

But they are not cases of oto-sclerosis. Inflation is useless because the actual causative factor has not been found. This is particularly so in those patients who state that, at times, their hearing is better, especially if they get a change of climate.

In making a diagnosis and prognosis of progressive deafness, there are many definite factors which are worthy of attention. The history of the patient is of prime consideration. Heredity, syphilis, the hearing during pregnancy, the history of intercurrent infections, the longevity of the case, must all be taken into consideration. I consider the occurrence of syphilis of great importance. It occurs often and when one least expects it. A recent case will illustrate this point:

Mrs. W, the wife of a physician, is suffering from a rapidly progressive form of deafness. The hearing in the left ear is gone. There is a residuum of hearing in the right ear. She can hear a loud voice near the ear. She hears fairly well with an electric phone on this ear. The drum looks normal. The Eustachian tubes are markedly stenosed and bougies can only be passed through the right one under direct inspection with the naso-pharyngoscope. I asked the doctor whether she had had a blood test taken. He informed me that she had had three or four taken, all of which were negative. However, I was not satisfied and had Dr. Winslow take one in our laboratory. The report came back 2 plus. The serum was sent to another laboratory and the same report came from Dr. Sondern. I expect to corroborate the diagnosis by spinal puncture. One can readily see that all the local treatment in the world will not cure this young woman, as long as the syphilitic taint remains in her body.

Among other factors of great seriousness are the intercurrent or chronic infections which give rise to a continuous irritative process. Whether this is accomplished through the formations of toxins or through local infectious processes, is of little moment. What is of consequence is that the elimination of these infections have a marked salutary effect on the deafness. Among the most important of these are the recurrent infections of the nose, throat and naso-pharynx, but one must not forget that the digestive tract is often at fault. The blood picture will often give a clue as illustrated in the following case:

Mrs. P—is markedly deaf, particularly in the right ear. Examination of the drums does not show marked retraction on either side although there is a slight amount of rigidity present. This is complicated by slight relaxation in certain parts. The patient has a marked stenosis of both Eustachian tubes, the right one being almost

impassable with the Eustachian applicator. She has been catheterized a number of times with the result that there has been a hyperinflation of both ears. Examination of the naso-pharynx, with the naso-pharyngoscope, revealed a decidedly polypoid enlargement of the posterior tips of the inferior turbinates on both sides, which no doubt was causing the irritated condition of the mucosa of the Eustachian orifice. That catheterization was the very worst thing that could be done in a case like this is self-evident. The only proper treatment to pursue is to get down the congestion around the Eustachian orifices without operation on the polypoid turbinates, if possible, on account of the age of the patient. She had been advised to have her tonsils removed. They are small and do not seem much diseased, but cultures taken from both tonsils shows a non-hemolytic streptococcus. A vaccine was made from this organism which will be administered. Wassermann test was negative. Her blood was chemically analyzed. It showed a marked increase in urea content.

In this case we see three important factors—the polypoid tips of the inferior turbinates, the infected tonsils and the altered blood chemistry. It is my opinion that the direct irritation from the tonsils may be overcome with the vaccines and that the polypoid condition of the inferior turbinates is a manifestation of some internal derangement which can be corrected by medical means. My advice to her was to place herself in the hands of some good internist who could raise her body resistance. I was more persuaded of the necessity for this when I found that local treatment of the nose and throat, with dilatation of the tubes but without direct treatment of her ears greatly improved her hearing.

That tonsillar infections, directly or indirectly, are responsible for diminution in hearing has been proved many times. Some eminent otologists feel that these organs are the cause of the trouble in almost every case or, to put it another way, any sign of a diseased tonsil is prima facie evidence that they are the cause of the trouble and should be removed. Oftentimes patients suffering from what may be called irritative deafness will at first complain of a stuffy feeling in the ears with a dullness in hearing, but tests show that the hearing is normal. A self-evident case is the following:

Mr. H—complained of a stuffy feeling in both ears with partial deafness. Tests showed very plainly that there was no diminution of hearing. Examination of the throat revealed very large and diseased tonsils. Wassermann test was negative. The Eustachian tubes were markedly congested, but the ears could be Politzerized without any difficulty.

This is another case where inflation of the ears would have resulted in disaster. I have no doubt that the tonsils are the cause of the trouble and have advised him to have them removed.

No less an authority than Dr. Francis P. Emerson, of Boston, states that he feels that the tonsils are directly responsible for progressive deafness in the majority of cases. He states: "—in every case of progressive deafness from chronic secretory otitis media, there is a primary focus, which is constant for that individual. This may become quiescent, but in the interval it remains as a low-grade process, subject to acute exacerbations. This point I wish to emphasize, for until the primary focus is drained and cured, such exacerbations are a constant menace to the Eustachian tube and middle ear. The course of the disease will be from bad to worse, and a little more rapidly if inflation is practiced as a routine." Again: "The tonsils in their relation to local and systemic disease are only beginning to be appreciated. * * * In many cases where the tonsil is the active cause of deafness, free pus will be demonstrated on one side * * * particularly in those cases showing general toxemia * * * Such cases are attended by a low-grade pharyngitis. In the writer's experience the extension to the Eustachian tube is more often by continuity and through the lymphatics and blood stream along the posterior pillar. The resulting low-grade process can be seen not only on the lateral wall, but in the epi-pharynx and over the surface of the posterior nares as well as in the mouth of the tube." In a recent letter to me Dr. Emerson says: "For the last two years I have been led to modify my idea of conditions in the naso-pharynx, and today I am of the opinion that a very large amount of the primary trouble starts in the tonsil, which is followed by a secondary pharyngitis, a blocking up of the back of the nose and involvement of the posterior ends and that the sinuses are of less importance in regard to hearing, although, of course, anything that keeps up a pharyngitis has a decidedly bad effect on the hearing."

I do not agree with Dr. Emerson in all his conclusions. Yet there is no doubt that the elimination of direct etiological factors is far more important than the routine inflation of the ears. The importance of systemic infections, from whatever source, is still not fully recognized. This statement leads me to another important point.

It is surprising to note in how many cases of deafness the posterior tips of the inferior turbinates are at fault. When the tips become polypoid, they overhang the Eustachian orifices and cause symptoms by direct irritation. The statement that such posterior

tips should always be removed is dangerous, for I am of the opinion that their enlargement is often due to some systemic irritation. Such a condition was present in the case cited a moment ago in which the posterior tips were polypoid and where an attempt has been made to diminish their size both by systemic and local treatment. It may be illustrated again in the following case:

Dr. A—complains of having marked diminution in hearing in both ears, particularly the right, for the past three years. There seems to be no causative factor such as syphilis. He had his tonsils removed, after which his hearing became worse. He has undergone various treatments, particularly inflation of the ears through a catheter, with the result that his hearing has not been improved, in fact, has diminished. Examination of his ear drums show that there is very slight retraction. Otherwise both drums look fairly normal with the exception of a small adhesion extending from the handle of the malleus toward the postero-inferior angle of the drum in the left ear. The amount of vibration of both drums is good. Hearing tests show that the hearing is better in the right ear than in the left. The lower forks are not heard in the left ear, but the higher forks are heard in both ears. Rinné negative. Whisper in the left ear is heard at fourteen inches. Voice and whisper normal in both ears. Such tests indicate that the hearing is fair in both ears, but the doctor cannot discriminate important sounds when using the stethoscope. In other words, his interpretation of sounds is faulty. In such a case it matters not what our hearing tests show, but it matters a great deal how the patient feels.

Examination of the naso-pharynx is interesting. By means of the naso-pharyngoscope, on both sides, one can see an interesting picture. On the left side, the opening into the Eustachian tube is fairly well marked. There are a number of discreet lymphatic glands on the promontory of the tube so that the tube is considerably narrowed. On the right side there are also glandular excrescences and there is a polypoid condition of the posterior tips of the inferior turbinates on both sides. Both tubes are slightly stenosed, but if the Politzer bag is used, inflation can readily be obtained in both ears by the slightest touch on the bag. This indicates that both tubes are wide open at the present time, and that any air that is forced into the ears under undue pressure will surely result in harm. If one were to catheterize very frequently, there is no doubt that complete deafness would result before very long. In other words, the only treatment that a patient of this kind needs is proper attention to the Eustachian orifices, and the polypoid tips of the

turbinates. This cannot be accomplished solely by local treatment. A systemic condition is also at fault, particularly a high blood pressure.

I have attempted to impress you with the fact that any etiological factor, whether it be localized in the naso-pharynx, nares or throat, or be general, has a direct bearing on hearing acuity, mainly by its action on the Eustachian tube. In the subacute conditions we frequently see the engorged tube, which can readily be shrunken and proper instruments passed through it. But we are more concerned with the chronic pathological states which give rise to intra-tympanic pressure. Certain tubes show a swollen, congested orifice with a pin-point opening. Other openings have a slit-like appearance. Sometimes the lips open easily. I have seen every variation from the atrophic tube, with glazed, glistening mucous membrane and a wide-open mouth to the hypertrophied tube with a mouth so tightly sealed that under no circumstances could proper atmospheric pressure be maintained in the middle ear. Sometimes a pathological condition will not be encountered until one reaches the isthmus of the tube, where he will find a definite stenosis. The variations in patency must be closely studied, and an interpretation of gurgling sounds, whistling sounds, sucking sounds and mucoid sounds, made so that one is able to follow the proper course of treatment.

I shall not attempt to go into detailed description of the treatment of deafness because each case is a law unto itself. Again let me impress you with the fact that it is high time that we recognized that insidious chronic affections of the ear are often caused by general toxic processes in the body.

A proper classification of treatment may be indicated as follows:

1. The elimination of the general systemic irritant such as syphilis, rheumatism, gout, blood diatheses, and any toxic process.

2. Attention to nose and throat abnormalities which might give rise to chronic nose and throat inflammations.

3. The proper treatment of the naso-pharynx such as removal of adenoids, freeing the adhesions in the fossa of Rosenmuller, the eradication of glandular excrescences on the Eustachian tubes, the proper treatment of polypoid tips of the inferior turbinate.

4. The direct treatment of the ears. For this to be accomplished one must differentiate between the hypertensed drum (commonly called O.M.C.C.) and the hypotensed drum (the relaxed drum).

A. In the former class of cases, the Eustachian tube is mainly at fault. Treatment should tend toward dilatation of the tube with proper applicators, sounds and bougies which can enlarge and medi-

cate the tube. After dilatation, the ear may be gently inflated, either by Politzerization or catheterization.

B. In the latter class, the hypotensed drum, an attempt must be made to contract the drum, which I have been able to do, in some cases, by the proper application of cantharides collodion. Inflation of such ears will make them worse instead of better.

5. There are certain cases of advanced deafness which cannot be relieved by medical means. To my mind such cases have been utterly neglected or mistreated. The physician should now turn to the psychological treatment of his patient. Mental reconstruction is what these patients need, so that they can regain sufficient confidence in themselves to again become important members of society. The treatment should consist:

A. In advising the patient, in the most sympathetic terms, of the nature of his trouble, of the uselessness of attempting to relieve it by medical means. Such a patient should be told that the future is full of promise if he will follow your advice.

B. The patient should be instructed to take up lip-reading at the earliest possible moment. He must be informed that learning lip-reading is like learning a new language, that it takes time and patience, but that the results have proved more than worth while.

C. The patient should be advised to join one of the various organizations which are attempting to mentally reconstruct the deaf. In New York, ten years ago, we organized the New York League for the Hard of Hearing for that purpose, and in Philadelphia Miss Flora E. Kinzie has been doing similar excellent work at the Philadelphia Speech Readers Guild. An American Association for the Hard of Hearing has been formed during the past year to carry on the work throughout the country. Leagues have been established in nearly twenty cities up to date.

Even as short a time as five years ago, a paper of this kind would have been listened to with casual interest. But the laity and the medical profession are beginning to awaken to the fact that the deafened form a large part of our communities and that their needs must be studied more thoroughly. We are forming a new and more intelligent conception of the problems of the deaf with the result that the future is full of promise. Deafened individuals are beginning to rouse themselves, too, which means that they will discuss their problems more freely and, perhaps, they themselves will be the ones to guide our future steps in their care.

2178 Broadway.

A RADICAL MASTOIDECTOMY FOLLOWED BY FORMATION OF A FALSE MEMBRANA TYMPANI AND NORMAL HEARING.

DR. LAWRENCE G. DUNLAP, Anaconda, Montana.

Consultation of the literature reveals several references to reports of radical mastoidectomy and resulting improved or normal hearing since Dr. Turch did the first radical operation in 1758 and reported it in 1761. However, a careful search of case records and papers on the subject is rewarded by only two reported cases of the formation of a false tympanic or manumetric membrane. J. M. Smith[1] reported a case of a girl aged 15, discharging ears 14 years, hearing spoken voice at two feet and a C^1 fork faintly heard in the air. Double radical mastoidectomy was performed a month apart, both ears dry about eight weeks after operation; hearing improved to, watch 12 inches, acoumeter 20 ft., whispers 20 ft., spoken voice 35 ft. in each ear and the formation of a secondary or manumetric membrane in the left ear (no description of membrane given). Case 2 by the same author was a female aged 17, discharge 15 years, radical mastoidectomy, ossicles destroyed, secondary skin graft 10 days later, ear dry in eight weeks and a secondary or manumetric membrane found here almost closing over the cavity (no further description of the membrane given); hearing unusually good, C^1 fork several seconds in the air, watch 18 inches, whispers 20 ft., acoumeter 20 ft., spoken voice 35 ft. Patient notices practically no difference in hearing between the operated and normal ear. The purpose of this report is to describe a third case in detail and offer some considerations relative to the secondary or manumetric membrane.

On December 19, 1919, Mrs. M. N., aged 24, married, 2 para, walked into the office very dizzy. This is her first time out of bed in 16 days. She states that she has had a discharging, foul smelling, deaf left ear since an attack of diphtheria at the age of three. Has had recurrent attacks of left-sided otalgia and been advised repeatedly to submit to a mastoidectomy. Sixteen days ago she gave birth to a still-born baby. On the eighth day of puerparium the left ear began to pain severely. The obstetrician gave her coal-tar products to relieve this pain and was partially successful. How-

*Read before the Silver Bow County Medical Society.

ever, today the pain and dizziness are too severe to continue such treatment. Heart trouble for some time. Tonsil operation at the age of three.

Examination revealed some necrotic teeth and infected gums, and absence of tonsils. At the tonsil operation most of the pharyngeal constrictor muscles were removed with the tonsils, resulting in a scarred pharynx and greatly constricted naso-pharyngeal opening. In the right naris is a low ridge along the vomer crest. Both middle turbinates are hypertrophied. The right drum is scarred and retracted, but she can inflate the Eustachian tube. The hearing test is as follows:

AD—Whispers, 6 ft.; Voice, 20 ft.; OC C^1 Fork, 10 sec.; AC., 5 sec.; Weber, 10 sec.; Rinne, Neg.; High Tone Limit, C_3 Long.

AS—Whispers, 4 ft.; Voice, 20 ft.; OC C^1 Fork, 11 sec.; AC., 8 sec.; Rinne, Neg.; High Tone Limit, C_3 Long.

The left drum is destroyed down to the annulus tympanicus; canal is filled with thick, foul smelling pus; no part of the malleus visible. There is a small polypus hanging down from the attic and extreme tenderness of the left mastoid over the antrum and tip extending down the sterno-mastoid muscle. No nystagmus or ocular deviation. Temperature normal, lungs normal. Double mitral heart murmur. The patient is a very sick, emaciated, nervous woman. No urinalysis because of lochia. No blood count made.

An immediate left radical mastoidectomy was advised and performed under ether two hours later. Operative notes were: a sclerotic mastoid cortex, cheesy cholesteatoma filling antrum, attic and region of annulus; necrotic incus and apparently a remnant of the malleus removed; radical mastoid finished by curettage of Eustachian orifice; plastic on the posterior canal wall, turning flaps to partially fill the mastoid cavity; posterior wound completely closed with a subcuticular and 5 catgut sutures; pack in the canal and cavity; operating time 52 min.; anesthetist noted no facial twitching, but on completion of operation the face is slightly drawn to one side and the left eye does not close. Dr. R. C. Hall, anesthetist; Dr. A. J. Willits, F.A.C.S., assistant.

The husband was told that if the patient survived the shock of the operation in her present depleted condition, she would at least be quite deaf in the left ear, from removal of the ossicles, and the ear might remain moist as the attempt to close the Eustachian tube too often is a failure. This was a frank prognosis consistent with the grief and difficulties accompanying such operations. Conval-

esence was uneventful. On the fourth day the patient was relieved of a slight dizziness by a cathartic. The facial paresis was less marked. Only a slight moisture in the left ear. She was out of bed on the seventh day and home on the tenth day with the ear canal clean and dry and the mastoid wound healed by first intention. High frequency current was used to stimulate the paretic facial nerve and motion returned in the face, forehead and lower jaw on the seventeenth day. A week later the face appeared normal; patient can wink and wrinkle nose and on the twentieth day the patient offered a surprise by hearing whispers at 8 ft.; on the twenty-sixth day she heard whispers at 20 ft., and continues to hear better over a year later.

Knowing that she had no drum or ossicular chain, I examined her to find the cause of this phenomenon and found that since removing the clolesteatoma and rendering the middle ear clean, a white, shiny epidermized membrane has grown over the whole inner wall and no doubt resting on the stapes, acts as a normal ear drum. The patient has gained in weight and is improved in general health since the operation.

On March 8, 1921, hearing was:

AD—Whispers, 7 ft.; OC., 15 sec.; AC., 8 sec.; Weber, 12 sec.; Rinne, Neg.; High Tone Limit, C_4 long. AS—Whispers, 66 ft.; OC., 15 sec.; AC., 12 sec.; Rinne, Neg.; High Tone Limit, C_4 very long. Left external canal, middle ear and mastoid cavity continuous. Inner tympanic wall covered by a membrane white, shiny, slightly transparent with a normal light reflex resembling a normal drum. This membrane extends continuously over the whole middle ear cavity, blends with the epithelial lining of exactly the same type which covers the mastoid cavity and passes over insensibly into the skin of the external canal wall which covers the facial ridge and mastoid cavity floor.

Theoretically, even in the presence of a secondary or manumetric membrane, the patient should hear poorly, at least not normally, because of the absence of the malleus and incus from the ossicular chain; however, this patient continues to upset theory. Our conception of the ossicular chain is that it acts as a "step up" transformer transposing the mass vibrations of the lower tones in their transmission through the drum membrane into molecular vibrations to the perilymph, 778 times[2] increased in intensity. The absence of the double fulcrum system of ossicles precludes such transformation in this case. Of course, sound vibrations are known to reach

the labyrinth through fenestra cochlea (round window) as the function of the ear is not altogether destroyed in otosclerosis or fixation of the footplate of the stapes. However, this small amount of hearing ordinarily so transmitted does not account for the hyperacuity of hearing in the case under consideration. The hyperacusis cannot be credited to hystery or neurasthenia, as it is unilateral and these are absent. Is not the excellent hearing due to a sounding board action of the smooth mastoid cavity? I offer this as a suggestion. The recent tuning fork test results in a negative Rinne and markedly decreased air conduction as compared to a normal hearing ear with bone conduction increased.

The abnormally keen hearing, more than twice that of the sound ear, causes no discomfort, the reverse being true, since the patient hears better than her normal companions, after being practically deaf on the left side since childhood. Will the good hearing persist? This, only time can tell, but as it has continued to improve for fifteen months, and there being no granulation tissue in the large cavity consisting of external and middle ear, antrum and mastoid cavity, and as the scar tissue already formed is probably mature and permanent in six to nine months, there is reasonable reason to believe the hearing improvement will be permanent and enduring. The physiological law laid down that,[2] "anything that interferes with the normal tension existing between the membrana tympani, ossicles and the contents of the oval window will cause tinnitus and deafness," may be considered proved by this exception to the rule.

The two quoted cases of manumetric membrane formation were not cholesteatomatous while this one was typically so, hence the presence or absence of cholesteatoma is evidently of no consequence in such membrane formation. The Eustachian tube is closed because the patient cannot Valsalvalize herself and the operative cavity is bone-dry and glistening since the first week after operation. That this result is merely chance is borne out by the poor prognosis given at the time of operation, which was the anticipated result. Again Esculapius is correct, "We dress wounds—God heals them."

Lawrence G. Dunlap, S.B., M.D.

BIBLIOGRAPHY.

1. The Radical Mastoid Operation: Indications, Technique and Results, Dr. J. Morrissette Smith, New York City, THE LARYNGOSCOPE, 28, 1918, p. 587.

2. BALLINGER, W. L.: Diseases of the Nose, Throat and Ear. 1914.

NATURE'S RADICAL MASTOIDECTOMY—A CASE REPORT.

Dr. L. W. Morsman, Hibbing, Minn.

Case No. 2441, male, forty years of age, occupation, traveling salesman, came to my office on September 22, 1920, on account of pain in the left ear of four days' duration. Patient gave a history of measles in childhood, following which the left ear discharged off and on until he became of age. After this he had only occasional discharge and earache to date, and stated that he had had the ear washed out by a doctor on the average of once a year. History was otherwise negative, including negative history of lues.

Ear, nose and throat examination was negative except that the left ear was filled with a mass of debris and pus with a necrotic odor. A large amount of this material was removed mechanically and the ear then thoroughly cleaned with alcohol and boric solution. A large, comparatively smooth cavity was found, connecting the middle ear, mastoid antrum and the external ear, as though a radical mastoid operation had been performed. The membrana tympanica, including the annulus tympanicus, was absent, as well as the malleus and incus. The cavity was intensely inflamed and in a septic condition.

The ear was cleansed daily with probe-tipped pledgets saturated with pure grain alcohol containing four per cent boric solution, following which, the cavity was packed with wick drain saturated with equal parts of compound tincture of benzoin and olive oil. The condition readily yielded to treatment and the two or three areas in which bone necrosis was still taking place promptly healed.

The ear now presents a clean scar-lined cavity, quite smooth, which has all the appearances of as neatly performed a radical mastoidectomy as could be accomplished with the electrically driven burr, with the exception that the floor is somewhat too high for proper drainage of the hypo-tympanum. The Eustachian tube is blocked to the passage of air by the Politzer method.

The patient stated that no operation had ever been performed upon his ear, and there is no doubt that the long-continued septic necrosis had, as it were, performed a typical radical mastoidectomy.

203 Congdon Bldg.

NASAL STATUS IN RETROBULBAR OPTIC NEURITIS.*

DR. THOMAS J. GALLAHER, Denver.

The present article is not intended to be a comprehensive presentation of the subject, but rather, a limited one.

While for many years it has been recognized that disease in the paranasal sinuses may produce affections of the eye, yet it is of comparatively recent date that critical study has been made of the relation of the posterior sinuses to the optic canal and optic nerve.

It is surprising that so few cases of retrobulbar optic neuritis, which have been relieved by nasal operations, have been reported by rhinologists, for it is certain that most of them have met with such cases.

Every case showing sudden blindness or diminution in vision should be referred at once to the oculist for a most careful examination of the eyes. The condition of the blind spot and the presence or absence of central scotoma for colors are essential to the proper diagnosis of this affection.

Once the diagnosis of retrobulbar optic neuritis is made, the rhinologist should be consulted and the proper operative nasal procedure instituted immediately. A patient in this condition is truly in a crisis, for upon proper diagnosis and most prompt surgical interference, his or her vision depends.

This certainly holds good in a vast majority of cases, while occasionally a spontaneous recovery occurs, but it is not to be depended upon.

The examination should be made with all possible haste and not a single day unnecessarily lost. Atrophy of the optic nerve may occur in fifteen to twenty days, which may result in permanent diminution in vision or blindness.

The general and blood examinations can be quickly made. All possible sources of focal infection should be eliminated, such as the apical abscesses of the teeth, infected tonsils, etc. The diagnosis is not difficult and is characterized by sudden diminution or loss of vision, accompanied by central scotoma for colors, the disc and fundus being normal.

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*Read before the Mid-West Section of the American Laryngological, Rhinological and Otological Society, Feb. 26, 1921, at Colorado Springs, Colo.

The differential diagnosis of optic neuritis from systemic causes, amblyopias, etc., should be made by the oculist. Optic neuritis from systemic conditions may exist in connection with retrobulbar optic neuritis. These conditions, I am convinced, must occur in scarlet fever and other exanthematous diseases in which there is a profound systemic infection accompanying local nasal infection.

It must be borne in mind that disease of the pituitary body also may produce similar conditions. The X-ray will be found very useful in giving the anatomic outlines and assisting in eliminating the possibility of pituitary disease.

The relation of the posterior ethmoid cells and sphenoid to the optic nerve of the same side, and to that of the opposite side, is indeed interesting and quite variable.

These sinuses may not only be in close relation with the optic nerve of the same side, but extend beyond the middle line in close relation with the optic nerve of the opposite side. It is therefore possible that either of these cavities may produce double optic neuritis, a point of practical importance. The optic nerve may traverse the cavity of the posterior ethmoid cell. Hyperplasia of the membrane, lining the posterior sinuses is the most frequent cause of this affection, with, or without, suppuration. Malignant disease may be a cause. Syphilis, as proven by the cases reported, must be a very rare cause.

If suppuration is present, the nerve may become involved by direct extension to the sheath or by the toxines. The low grade of inflammation in the hyperplastic tissue extends to the neighboring parts, involving the back of the orbit, and particularly causing a swelling of the periosteum in the optic foramen which results in a compression of the optic nerve.

The central scotoma is produced by the peripheral pressure causing a compression of the delicate fibres in the center of the nerve, which supply the macula. Hence it is one of the first symptoms.

The fundus shows no vascular changes, due to the anatomic fact that the vessels pierce the sheath back of the orbit, anterior to the optic canal.

We believe that disease in the Antrum of Highmore, either of hyperplastic or suppurative type, may also produce this affection by involving the adjacent ethmoid cells, which in turn may affect the optic nerve in the foramen or in its course. Any nasal defect or systemic condition which produces nasal inflammation, predisposes to this affection.

In the hyperplastic type, the examination of the nose may show apparently a normal nostril. It should be emphasized that no pus is present. The absence of pus is indeed too often taken as proof that the nasal condition is normal.

The question immediately arises, "Is it possible for us to absolutely determine in all cases that a nostril is normal by visual inspection?"

It is not possible, because we cannot tell the condition of the membrane lining the cavities.

If, therefore, after a definite diagnosis of retrobulbar optic neuritis has been made, and no pathological condition can be demonstrated in the nose, using all the means at our command, nevertheless it is entirely justifiable to open the posterior ethmoids and sphenoid.

We have been accused as rhinologists of performing too many operations in the nose, but the results attained, in operating under these circumstances, are so brilliant, that even the most critical must be convinced of the wisdom of this course.

It is infinitely better, if needs be, to sacrifice the middle turbinate, the ethmoid cells and open the sphenoid, rather than to jeopardize our patient's vision.

In many cases abnormalities are found, such as deflection of the septum, a demonstrable hyperplasia of the middle turbinate, with its usual accompanying hyperplastic ethmoid cells.

The septum and middle turbinate should be critically examined throughout their entire length. Shrinking the tissues about the turbinate and moving it over with a spatula, may demonstrate that it is not normal.

Suppuration in the posterior sinuses, producing optic neuritis, should be promptly dealt with surgically.

The operation performed by most rhinologists consists in a correction of the deflected septum, if it exists; removal of middle turbinate and exenteration of posterior ethmoid cells, and cutting down the face of the sphenoid. Many rhinologists think it wise to make a complete exenteration of both the anterior and posterior cells, in addition to establishing sphenoid drainage.

As a prophylactic measure, it may be stated that a correction of all nasal defects early in life, is advisable.

As a digression, we beg to say that we believe that paralysis of the trunk of the facial nerve, as it passes through the Fallopian canal, may be produced by a hyperplastic condition in the middle ear, or

in the adjacent mastoid cells, in the same manner which cause the retrobulbar optic neuritis.

A swelling of the periosteum, in both instances, is probably the cause of injury to the nerves. Both of these nerves are in a similar relation to adjacent air cavities.

In many cases where early diagnosis has been made and thorough nasal correction has been attained, the restoration of vision to a greater or less degree is indeed remarkable. And so, as rhinologists, let us intelligently co-operate with our esteemed ophthalmologists to the end that we both may be the means of preserving this most precious sense.

Let it be not said of either of us, that through a lack of interest or a thorough investigation, that a single patient has been doomed to blindness through any acts of omission or commission.

I desire to report four cases illustrating different types of this affection:

Case I. A woman, age 39, married, was seen October, 1912. For two years previously she had suffered from transient amaurosis monthly, lasting from a few minutes to a half hour, with a continuous floating speck before the right eye.

The custom which was in vogue then to correct these defects by proper glasses was carried out during that time by her oculists. The vision in the right eye suddenly fell to counting fingers at three feet, October, 1912. The central scotoma was not reported upon by her oculists.

The nasal examination showed an S-shaped deflection of the septum obstructing the left nostril in front and the right nostril posteriorly. The left middle turbinate was hypertrophied and the membrane on both sides presented the appearance of vasomotor rhinitis.

Immediate nasal operation was advised and performed, notwithstanding one of her oculists advised against operation.

The deflection was corrected, middle turbinectomy done, exenteration of the anterior and posterior ethmoids, free opening of the sphenoid. Improvement in vision for fingers to six feet occurred within forty-eight hours and in two weeks fingers could be seen at twenty feet.

Four months R.V. 5/30 mostly, to 5/15 partly. Vitreous then showed one large mass, several thin plaques and considerable dust.

Five years later R.V. 5/7. Best ever obtained was 5/6, and vitreous showed only one tiny opacity.

Had this patient been operated upon two years previously she would probably have avoided all of this trouble, and it is interest-

ing to note the progress made in the treatment during the last ten years.

Case II. Woman, age 45, married, seen October 10, 1918.

She had been afflicted with periodic dimness of vision in left eye for five months. She was told the trouble was due to some systemic poison, but no definite method of procedure was advised or instituted by her oculists.

She was referred by me to Dr. George F. Libby, who returned the following report: "Disc margins obscure, nerve heads hyperemic and slightly swollen. No scotoma present. Diagnosis moderate optic neuritis L.V. 5/5 mostly."

The nasal examination showed septal deflection to the left, causing great obstruction.

Submucous resection of the septum was now done. Improvement immediately followed, disc margins became sharper, hyperemia disappeared with vision of 5/3 mostly in each eye.

Seven devitalized teeth were extracted, two showing streptococcus viridans.

Six weeks after this the vision in the left eye suddenly fell to 5/10. Disc normal with distinct central scotoma for colors. The prompt diagnosis of retrobulbar optic neuritis was made.

The nasal operation was performed at once, which included middle turbinectomy, exenteration of the anterior and posterior ethmoids and cutting down the face of the sphenoid on the left side.

In three weeks the vision had returned to normal and has remained permanent.

This case undoubtedly shows optic neuritis caused by systemic disturbance, which in a short time was followed by a retrobulbar optic neuritis.

Case III. Woman, age 35, married. Ocular examination made by Dr. George F. Libby, giving the following report, Dec. 24, 1920:

During past ten years intermittent blindness in the right eye which became permanent three months ago. In August, 1920, L.V. dropped to 5/30, rose to 5/7 partly in following three months under specific treatment.

The hematologist reported a slightly positive Wassermann by one of the refinements in the test. This was not corroborated and is open to question. I do not believe that there was any specific element in the case.

Central scotoma was present in the lately affected eye.

In November, both vitreous bodies were cloudy, discs red, fundus details obscure, but no intra-ocular hemorrhage, floating opacities in each vitreous. Old choroiditis lower quadrant in R. Small posterior polar cataracts.

Diagnosis of retrobulbar optic neuritis of probably four months' duration was made and the usual nasal operation was performed with the hope of improving the left eye and preventing total blindness.

In two months the vision has fortunately improved to 5/6, which we think will be permanent.

This case illustrates the fact that it is justifiable to perform the nasal operation even at a late date to preserve the remaining vision.

Case IV. Woman, age 38, married. Referred to me by Dr. George F. Libby, who made the ocular diagnosis of retrobulbar optic neuritis. Two years ago, blindness one-half hour in both eyes.

Vision much blurred in past ten days and has floating specks before eyes. Binocular central scotoma present. R.V. 5/30 partly, L.V. 5/30 mostly, fundus normal, Wassermann negative.

Nose showed deflection of the nasal septum to the right with synechia between the middle turbinate and septum posteriorly. The right middle turbinate was abnormally close to the external wall. The left middle turbinate was apparently hyperplastic.

Correction of the nasal septum and the usual operation upon both sides was advocated. As I was leaving the city the next day one of my confreres kindly performed the operation as advised.

Two diseased teeth were also extracted and a tonsillectomy performed later. Improvement followed in a very few days after the nasal operation.

Six weeks after the nasal operation R.V. is 5/4 partly and L.V. 5/4 mostly.

In this case complete restoration of vision was obtained by prompt diagnosis and immediate operation.

California Building.

CASE OF NASAL HEMORRHAGE (POST-OPERATIVE) REQUIRING LIGATION OF EXTERNAL CAROTID ARTERY.

DR. HERMAN B. COHEN, Philadelphia.

Mrs. Y. N., age 43, referred for nasal treatment to aid in the correction of a gastric affection. The past history is negative with the exception of a gastro-enterostomy in 1912, for gastric ulcer.

October 20, 1920, I did a Ballenger exenteration on the right side and a less radical exenteration on the left for bilateral hyperplastic (polypoid) ethmoiditis. The patient was discharged from the hospital on the 22nd. Coagulation test at this time was within normal limits and patient bled the usual amount after such a procedure.

On November 18, 1920, at one of her visits to the office, I noticed a sessile polyp in the region where the posterior end of the right middle turbinate had been. I removed it under a little cocaine and adrenalin and because the patient desired to go shopping I placed a small piece of iodoform selvidged gauze in apposition to the operated area. Within an hour the patient phoned that she was bleeding quite badly, whereupon I requested her to return to the office at once. The sight beggared description. Every attempt to control the bleeding was in vain, which included nasal and post-nasal packing. Finally, finger pressure through the posterior choana controlled the bleeding, but any relaxation of pressure resulted in more bleeding. With my finger in position above stated I took the patient to the hospital, where I had the kind assistance of Dr. Coates, and we again tried nasal and post-nasal packing. The blood showed no attempt toward clotting. Everything known to us was used for the control of hemorrhage and shock.

November 20, I removed the packing and that evening the patient had another severe hemorrhage requiring packing. After three days I removed the packing and that night she had another hemorrhage. The next morning the patient bled through the nasal packing, but not as severely as in previous hemorrhages. At 8 P. M. that night she had another hemorrhage and again the next morning, including bleeding into the pharynx. By this time the patient's condition was becoming alarming and that evening a transfusion was done, taking 500 cc. of blood from a donor, a nurse

at the hospital. Fifteen minutes after the transfusion, performed by Dr. Ravdin, the patient began to bleed again and showed signs of dying. We decided upon another transfusion the next day and ligation of the external carotid artery as the only thing remaining that might save the patient's life. This was done 24 hours later, and from then on the patient made an uneventful recovery. On the 24th of December the patient left the hospital in good condition.

The red cell count improved from 2,500,000 on November 24th, to 3,900,000 on December 21, the hemaglobin remaining unchanged at 45%.

A study made by Drs. Bartlett and McKitrick of St. Louis of 105 cases of ligation, 15.25% were done for secondary hemorrhage. Of those, that interest us as rhinologists, two are reported, viz: one by Dr. Thormer, in 1897, for nasal epistaxis, and the other by Dr. Lane for post-tonsillectomy.

"The mortality attending this treatment of secondary hemorrhage with its otherwise practical certainty of a speedy fatality, is no greater than the general mortality which has followed the ligation of the large common carotid in many hundreds of instances in the treatment of the greatest variety of pathological conditions."

1301 Spruce Street.

NEW TONSIL INSTRUMENT.

Dr. Henry M. Goodyear, Cincinnati, Ohio.

The accompanying cut of this instrument is sufficient to explain the general mechanical construction. As to application, the instrument is applied to the tonsil after the fashion of any tonsil enucleator, built on the plan of a permanent ring fenestrum.

The handle is so placed that the force is applied at right angles to the point of application to the tonsil.

After engaging the tonsil through the fenestrum, the finger piece is raised, locking automatically, and the final enucleation is completed by slowly turning the ringed screw at the top.

The dull ring separates the tonsil from behind forward, following the line of extra capsular clearage, and bluntly dissecting the tonsil from its bed.

Over one hundred cases have been done with the last model of this instrument, and it has been found practically impossible to injure the tonsil pillars or the superior pharyngeal constrictor muscle back of the tonsil; the tonsil coming away in its capsule, clean of muscle fibers. There is a minimum of bleeding and in a few of these cases there was not sufficient bleeding to necessitate the use of suction or a sponge.

I finished the first model of this instrument in 1917, and after having revised it many times in getting the proper proportions and principal and having faithfully tried it in the field of operation, I believe I can conscientiously recommend it as an instrument simple and durable in construction, which gives the minimum of bleeding and trauma, with a complete and simple enucleation of the tonsil.

It is especially well adapted to use of general tonsillectomies in children.

8 East Eighth Street.

CASES OF POST-NASAL CHOANAE MALFORMATION BY BONY OCCLUSION.

Dr. James C. Kirby, Boston, Mass.

The main idea of this paper is to enable a physician to diagnose this malformation by a few additional signs and symptoms. The operation in itself is practically the same as has been described by various authors in the past. The conclusions are drawn from a limited number of cases chiefly because of the rarity of the cases.

Minnie B.: Past history of nasal obstruction and considerable amount of discharge from nose; unable to blow mucus from nares; the only way the mucus could be obtained was by hending the head forward.

General condition: Health rather poor; patient very nervous at intervals; asymmetry of face.

Previous operations: Tonsils and adenoids. Later, secondary adenectomy. No relief obtained from nasal discharge and obstruction by these operations. The only relief obtained from the nasal discharge was by irrigation.

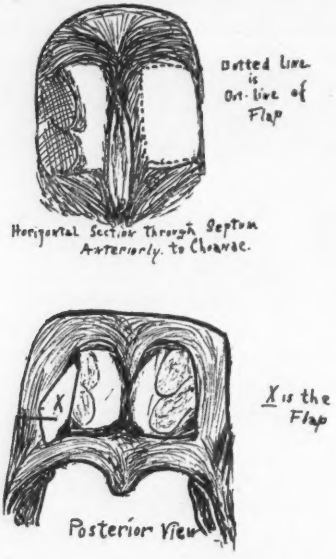
Examination: Bluish-white discharge, the consistency of mucoid material, from right nare. This had to be wiped away before view of turbinates could be obtained. Turbinates were pale and the size of atrophic turbinates. Obstruction to probe in nasal choanae of that side. View with mirror confirmed diagnosis. Throat, negative. Ears: Marked retraction of drum of right; tests gave evidence of catarrhal deafness of that side.

Operation: Submucous resection, and removal of malformation through this route; then a window flap of the mucous membrane, having its base at the pharyngeal side of the choanae, the idea of such procedure being that this flap later degenerates and the opportunity for granulations is less by this method. Rubber tubes, diameter about ½ inch, were then placed to keep the continuity of the opening. The orifice of the Eustachian tube could not be made out, as there was a band of tissue over it.

(Note: Be careful no ridge remains on the floor of the septum, because of the difficulty in entering the tubes, which enable the opening to be held patent.)

For three weeks following the operation, especially the first week, there was a marked increase of muco-purulent nasal discharge from the right side. The antrum was negative from the readings of the X-ray plates, which were confirmed later by puncture. The choanae opening remained patent for 1½ years. After that time, I lost sight of the patient.

Margaret D.: Age, ten years. History of malnutrition. Mother stated that child would not take breast long when a baby and always seemed to have a head cold.



Previous operations: Tonsils and adenoids. Later, adenectomy.

Same history as the previous case of no relief of discharge after these operations. At the time of my examination the discharge was of the same color and consistency and the appearance of the turbinates the same as in the previous case.

Operation: Submucous resection; window opening. Plate of bone obstructing the choanae was very thin and joined the vomer just 5 m. from the posterior border.

Post-operative treatment with tubes resulted in same history as previous case, namely: profuse nasal discharge, clearing up later under douche and spray treatment. Tubes should be inserted daily

and before being replaced should be sterilized and coated with vaseline. These tubes should be gradually left out, beginning at the end of the second week and increasing the time between each re-introduction, until at the end of the fourth week they may be eliminated entirely. The second week, in the case of Margaret D., the tube caused considerable irritation of the posterior tip of the interior turbinate, which resulted in granulations or polypi. The tube was omitted four days, then replaced. Child's health greatly improved, appetite recovered. Mother stated that it was a different child. In both cases the window flap degenerated and left a very little pedicule on the pharyngeal side.

CONCLUSIONS.

Nasal method by submucous resection route and window flap made of mucous membrane leave least possible ground for granulations.

The peculiar bluish color and consistency of the discharge and the anemic turbinates before operation are of value as an additional diagnostic aid in this condition of occlusion.

The only other condition where the turbinates have the same appearance as in the above cases is that of the tubercular turbinate.

The post-operative treatment varies in no way from that which has been mentioned in previous articles, but, in addition, the purulent nasal discharge which makes its appearance is in all probability due to the irritation caused by the tubes and simulates sinusitis.

With regard to the age of the patient operated upon, I think it is more advantageous to operate on children because first, it helps in the development of the child, and second, the occlusion, if it is bony, is very thin compared to that of an adult, which is more ossified. The disadvantage of operating on children is the limited space for operation, and the oblique angle at which the choanae openings are placed. In adults, these openings assume a more vertical position.

MEMBRANOUS OBSTRUCTION OF THE BRONCHI, CLINICALLY DIPHTHERITIC, WITH REPEATED REMOVALS WITH THE BRONCHOSCOPE—RECOVERY. APPARENTLY TWO NEW TRIUMPHS FOR BRONCHOSCOPY.

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Membranous obstructions of the bronchi, unless exfoliation takes place and spontaneously coughed up, will usually cause fatalities. Before the days of antitoxin we frequently found membranous obstructions in the larynx and even below the tracheotomy wound. It has been reported to me by a number of my doctor friends that they had cases where a cast of the trachea and bronchi had been coughed out spontaneously with the resultant cure of the child. In my experience, whenever a membraneous deposit sufficient to cause cyanosis formed below the tracheotomy wound it has invariably been fatal. I can recall several cases in which the membrane formed thus that had bronchoscopic removal been done probably I could have had several more lives to my credit. Until the case herein reported was encountered I gave up just as soon as these obstructions below the tracheal wound occurred. However, it seems that bronchoscopy has triumphed again and I am herewith reporting not only a successful removal of a membrane bronchoscopically, but also apparently another use for the bronchoscope.

Case Report: In the afternoon of January 12, 1921, I was asked by Dr. J. B. Acree to see a boy, four years, nine months old, who had very difficult breathing. On reaching the patient I got the following report:

The parents stated that the child had been sick for a week with high- intermittent temperature, sore throat and enlarged glands of the neck. The parents thought that the trouble might be diphtheria, but they failed to call a physician until January 11, when Dr. Acree saw the child and made a diagnosis of diphtheria and gave 10,000 units of antitoxin at 7:30 A. M., on this date. There were no other symptoms from the onset of the trouble except those mentioned until 2 A. M., January 11, when violent coughing set up and hoarseness developed. Dr. Acree saw the child again in the after-

noon of the same day and the symptoms were much improved. He saw the child again at 8 A. M., January 12, and administered 10,000 units more of antitoxin. He again saw the child in the afternoon of January 12, and, as it was very much worse, he decided that some means of relieving the dyspnea was immediately needed. When I arrived at the house of the little patient it was evident that we did not have time to get the child into the Isolation Hospital, but that immediate relief of some kind must be had. I decided that a tracheotomy was the operation of choice, which I at once carried out, with the result that in a few minutes the child was sufficiently recovered to send to the isolation ward.

The child entered the hospital at 6:45 P. M., first month, twelfth day, with temperature of 102° and pulse of 130°, and breathing

Tracheal wound completely healed and perfect recovery of boy as evidenced by his apparently perfect physical condition.

well through the trachea tube. The child spent a restless night with a great deal of coughing and, on the morning of the 13th, it seemed not to be getting sufficient air. I thought perhaps that the trachea tube that I had inserted the day previous was too small; so, therefore, it was decided to insert a larger tube, which was done about 9 A. M. It spent a restless day and night following this and on the morning of the 14th I was telephoned to that the child was very serious and to come at once. When I reached the hospital it was very evident that the child was in a serious condition. I removed the trachea tube and, with a little cocaine and adrenalin applied to the wound, I pulled the trachea forward and could see almost to the bifurcation and I concluded that the obstruction must therefore be

in the bronchi. The plan then occurred to me to attempt to look into the bronchi, as there was nothing else to be done, and to remove the obstruction, if possible, with forceps through the bronchoscope. With the presence and help of Drs. J. B. Acree and R. E. Hearne, and nurses, I at once passed a bronchoscope into the right bronchus and could see that it was almost completely occluded with a white, grayish membrane. It was at once grasped with forceps, and tube, forceps and the membranous obstruction all withdrawn together. This mass probably was almost a complete cast of the right main bronchus. I again inserted the bronchoscope, this time into the left bronchus, and found that it had the same conditions present. I seized the membrane, as on the right side, but, as this membrane was not so tenacious as the other side, I got only a small piece, but was fortunate enough to dislodge another larger piece which, while I was cleaning out the forceps, was coughed up into the trachea wound and removed by Dr. Acree with a pair of forceps. The same procedure then was carried out on the right side and I succeeded in getting a small piece, also dislodged another piece, which was coughed up into the trachea wound and removed by forceps. All these pieces and slugs were requested to be kept and a photograph and laboratory examinations were to be made. Unfortunately, in the excitement and under the stress of the situation, these pieces were destroyed and we have no laboratory report that the case was diphtheria. It was very gratifying at this particular stage of the game to note that the child breathed perfectly and that he was apparently no worse off for this trying ordeal, not the bronchoscopy, but the fight for air. The child got through the day fairly well, but toward evening it began again to have difficult breathing, much to our disappointment. By 8 o'clock P. M., I saw it would be necessary to attempt another removal of the membrane, which I did with the assistance of Miss Thomas and Miss Ross. The method of procedure was the same as in the morning and the child got immediate relief and went at once to sleep.

By the morning of the 15th, we had a repetition of the same conditions as on the morning of the 14th and, accordingly, at 8 A. M., with Dr. Acree present and the same nurses, I again removed the membrane. Again the child got immediate relief and had a very fair day. By 6 P. M., we had a repetition of the same difficult breathing and I again removed the membrane and again the child got immediate relief and went to sleep. It became evident that something else must be done or the child could not stand the exhaustion incident to this difficult breathing. As the case had been

diagnosed diphtheria before its admission into the hospital and, as the membranes kept reforming, we considered this sufficient evidence that we were dealing with a case of diphtheria without resorting to any laboratory tests. And, as our city did not have a laboratory in connection with the hospital, we felt reasonably sure that we were dealing with diphtheria; so, accordingly, agreeing with Dr. Acree that the child had not had enough antitoxin, we began its administration at 8:35 P. M., giving 20,000 units. All bronchoscopies were done without any anesthesia, except the first, in which I had previously used some local application of cocain to the tracheal wound in order to establish the location of the obstruction. The following table will show the administration of antitoxin, the removal of the membrane bronchoscopically, and will give some idea of the progress of the case. I shall leave out as many of the details as deemed necessary in order to make the report more concise:

At 6 P. M., I was telephoned for to come at once, as the child was again in very serious condition. On entering the room I was told by the nurses that I had probably come too late, as the child was almost dead. After a few convulsive movements the child appeared dead. A hasty examination revealed no respiration, no pulse, no heart beat and no evidence of life. The child looked like a cadaver.

Immediately I asked for a small bronchoscope and inserted it hurriedly into the right bronchus and through a thickly formed membrane. I called on the nurses to give artificial respiration, and at the very first effort it was evident that we were getting air into the lungs and within a few seconds we saw there was a chance of reviving the child. Artificial respiration was kept up for a few minutes and the color and respirations very much improved. Just as soon as I considered the child strong enough, I withdrew my tube through the membrane and seized it as I had repeatedly done before, withdrawing it. Repeating this in the other bronchus we soon had a perfectly breathing child, apparently little the worse for the trying ordeal through which it had just recently passed.

As there was no further difficulty in breathing, it was decided that probably the child had had enough antitoxin. At this time it became necessary to change nurses, as those already on the case were worn out from continuous efforts to relieve the child. Their place was taken by Misses Noonan and Wildt, who assisted in the following bronchoscopies and looked after the child until it was dismissed:

Date, 1921	Time	Temperature	Dose, Units	Given by—
1/15	1:35 P. M.	99	20,000	Dr. Purcell.
1/16	2:00 A. M.	Bronchoscopic removal of membrane by Dr. Purcell.		
1/16	2:00 A. M.		20,000	Dr. Purcell.
1/16	4:00 A. M.	100[4]	10,000	Miss Ross.
1/16	6:00 A. M.	Bronchoscopic removal of membrane by Dr. Purcell.		
1/16	6:30 A. M.	101[2]	10,000	Miss Ross.
1/16	10:25 A. M.	101	10,000	Dr. Acree.
1/16	3:30 P. M.	101[2]	20,000	Miss Ross.
1/16	6:00 P. M.	Bronchoscopic removal of membrane by Dr. Purcell.		

Date, 1921	Time	Temperature	Dose, Units	Given by—
1/16	11:30 P. M.		20,000	Dr. Purcell.
1/17	6:30 A. M.	100	No slugs.	

Date 1921	Time	Temp.	
1/18	10:30 P. M.	100	Bronchoscopic removal.
1/19	7:30 A. M.	99	No reformation of membrane.
1/20	11:30 P. M.	98[4]	Child broken out with intense eruptions.
1/21	2:30 P. M.	99[3]	Child restless; intense itching.
1/22	3:00 A. M.	99	Trachea tube removed.
1/22	9:00 A. M.	99[4]	Wound closed with adhesive; patient talks aloud.
1/23	12:00 Noon	97[6]	Tube out; breathes well; talks well.
1/23	4:00 P. M.	99	Patient dressed and up in chair.
1/24	8:00 A. M.	100	Dressed and walks about.
1/25	6:00 P. M.	98[2]	Walks about all day.
1/27	6:00 P. M.	99	Walks about all day.
1/28	4:00 P. M.	98[2]	Patient been playing all day.
1/29	7:00 P. M.	98[3]	Patient up all day.
1/30	8:30 P. M.	98[3]	Patient discharged cured; trachea wound closed.

Conclusions: 1. That in our opinion tracheotomy is the operation of choice in laryngeal stenosis due to membranous obstruction.

2. That stenosis from membranous formation below the tracheal wound offers hope of relief by use of the bronchoscope.

3. That if there is no laryngeal stenosis and no membrane within the larynx, bronchoscopic removal may be safely done through the mouth and, judging from the case reported, there should be little or no reaction from bronchoscopic removal through the trachea wound if gently and carefully carried out.

4. That we are making no attempt to justify the diagnosis of diphtheria which was made on clinical evidence alone.

5. That we felt amply justified in giving the large amount of antitoxin because up to the time of the beginning of its administration there were no evidences of improvement in the child's condition.

6. That in general, in case of even moderate cyanosis, unless its cause can be accurately accounted for, the bronchoscope is always indicated.

7. That the bronchoscope offers a new chance of relieving apparently hopeless cases of asphyxiation in children with tracheal or bronchial membranous obstruction.

STAMMERING—A BIBLIOGRAPHY OF THE PAST DECADE—1911 TO 1920, INCLUSIVE.

DR. C. S. BLUEMEL, Denver, Colo.

APPELT, A.: Fortschritte der Stotterer-Behandlung. Heilen und Bilden, 1914. Stammering and Its Cure. Lond., 1911.

ARONSOHN, O.: Der Psychologische Ursprung des Stotterns. Halle, 1914. Zur Psychologie und Therapie des Stotterns. Berl. klin. Wchnschr., 1911, xlviii, 88, 170-174.

ASH, E. L.: Stammering and Successful Control in Speech and Action. Lond., 1916.

BIAGGI: Prognosi Della Balbugie. Critica Med., Milano, 1914, iv, 1.

BLANTON, M. G.: What is the Problem of Stuttering? J. Abnorm. Psychol., Bost., 1918-19, xiii, 303-313. Also Quart. J. Speech Educ., Menasha, Wis., 1919, v, 340-350.

BLOCK, S.: More Common Speech Defects for the General Practitioner. Long Island M. J., Brooklyn, 1914, viii, 209-214.

BLUEMEL, C. S.: Stammering and Cognate Defects of Speech. 2 v., N. Y., 1913.

BRIAND, M., and PHILLIPPE, J.: Un cas de Bégaiement Paroxystique d'origine Émotionelle; Traitement; Guerison. Progrès Med., Par., 1917, 3 s., xxxii, 257-261.

BROWNING, W.: Etiology of Stammering and Methods for Its Treatment. Neurographs, Brooklyn, N. Y., 1915, i, 213-335.

BRUCE, H. A.: Stammering and Its Cure. McClure's Mag., N. Y., 1912-13, xl, 91-101.

BRUMMELER, M.: Vocal Treatment of Stuttering. Pedagog. Sem., 1918, xxv, 97-104.

BRYANT, F. A.: Influence of Heredity in Stammering. J. Hered., 1917, vii, 46. Questions and Answers About Stammering. N. Y., 1916. Some Speech Disorders and Their Treatment, Together With An Outline of Methods Used. N. Y., 1913. Stuttering and Its Treatment. Med. Rec., N. Y., 1913, lxxxiv, 614-619.

CADWALLADER, B.: Elimination of Speech and Voice Defects. LARYNGOSCOPE, St. Louis, 1915, xxiv, 986. Treatment of Stuttering and Stammering and Voice Defects Through the Science and Aid of Speech and Singing. Cleveland M. J., 1911, x, 1025-1030.

CARRIE, W.: Statistik Ueber Sprachgebrechliche Schülkinder in den Hamburger Volksschulen (Jan., 1917). Monatschr. f. Ohrenh. (etc.), Berl., 1918, lii, 178-187.

CARSWELL, E. R.: Cause and Cure of Stammering and All Other Speech Defects. Chicago, 1912.

CHARNLEY, C.: Experiences in the Treatment of Vocal Disorder. Boston M. and S. J., 1913, clxix, 833. Some Observations on the Conditions and Treatment of Stuttering. Boston M. and S. J., 1913, clxix, 824-828.

CHERVIN: De l'Intermittence dans le Bégaiement. Semaine Méd., Par., 1913, xxxiii, 253. Du Role de l'émotion en Pathologique Verbale; Bégaiement Blésité. Semaine Méd., Par., 1912, xxxii, 37-40.

CLAIBORNE, J. H.: Stuttering Relieved by Reversal of Manual Dexterity, with Remarks on Symbol Amblyopia. N. Y. Med. J., 1917, cv, 577.

COPLEY, F. B.: Good News for Stutterers and Other Defectives in Speech. Am. Med., 1920, xc, 32-33.

CORIAT, I. H.: Stammering as a Psychoneurosis. J. Abnorm. Psychol., Bost., 1914-15, ix, 417-429.

CREASY, H. M.: Psychopathology of Speech Defects. Quart. J. Speech Educ., Menasha, Wis., 1919, v. 6-11.

DA COSTA FERREIRA, A. A.: A Phisionomia Ocular dos Gagos. Arch. de Anat. (etc.), Lisb., 1915, iii, 65-67. Stuttering of Endocrine Origin. Arch. Méd. Belges, 1919, lxxii, 680.

DUNLAP, E.: Stuttering Boy. J. Abnorm .Psychol., Bost., 1917, xii, 44-48.

DUPUIS, L., and LEGRAND, A.: Le Bégaiement. Bull. de Laryngol., Otol. et Rhinol., Par., 1913, xvi, 147-167.

EDER, M. D.: Das Stottern, eine Psychoneurose und Seine Behandlung durch die Psychoanalyse. Internat. Ztschr. f. ärztl. Psychoanal., Leipz. u. Wien., 1913, i, 547-555. Also Tr. Internat. Cong. Med., 1913, sec. xii, Psychiat., pt. 2, 187-192.

ELDERS, A.: Heilung des Stotterns nach Gesanglichen Grundsätzen. Leipzig, 1911.

ERSNER, M. S.: Defective Speech and Some of Its Phases. LARYNGOSCOPE, St. Louis, 1918, xxviii, 416-421.

ESPEJO, L. D.: Algunas Consideraciones Sobre Tartamudez. Cron. Méd., Lima, 1918, xxxv, 191-198.

FLETCHER, J. M.: Etiology of Stuttering. J. Am. M. Assoc., Chicago, 1916, lxvi, 1079-1081. Experimental Study of Stuttering. Am. J. Psychol., Worchester, Mass., 1914, xxv, 201-255. Mental Imagery of Stutterers; An Examination of Certain Current Theories. J. Abnorm. Psychol., Bost., 1917, xii, 34-43. Speech Defects in Children. Am. J. Obst., N. Y., 1912, lxv, 176-186.

FOY, R.: Le Bégaiement; Comment il s'Etablit; Comment le Reconnaitre, le Prévenir et le Traiter. Enfance Anorm., Par., 1914, iii, 197; 279. Le Bégaiement; Nouveaux Essais Pathogéniques et Therapeutiques. Rev. Hebd. de Laryngol. (etc.), Par., 1913., ii, 545; 577; 609. Le Bégaiement Pathogénie et Traitement. Clinique, Par., 1914, ix, 266-268. Traitement du Bégaiment par le Ductophone. Bull. d'Oto-Rhino-Laryngol., Par., 1913, xvi, 342-349.

FREMEL, F.: Stottern und Fazialsphänomen. Wien. Med. Wchnschr., 1913, lxiii, 2207-2209.

FRÖSCHELS, B., and FREMEL, F.: Gehör und Sprache. Beitrag zur Aetologie des Sigmatismus. Arch. f. Exper. u. Klin. Phonet., Berl., 1913-14, i 205-214.

FRÖSCHELS, E.: Lehrbuch der Sprachheilkunde. Leipz. u. Wien., 1912. Stottern und Nystagmus. Monatschr. f. Ohrenh. (etc.), Berl. u. Wien., 1915, xlix, 161-167. Tonisches Stottern Ausgelöst Durch Explosions Luftdruck. Monatschr. f. Ohrenh. (etc.), Berl. u. Wien., 1914, xlviii, 1291.

FRÖSCHELS, E.: Ueber das Wesen des Stotterns. Wien. Med. Wchnschr., 1914, lxiv, 1067-1076. Also Beibl. z. d. Mitt. d. Gesellsch. f. inn. Med. u. Kinderh. in. Wien., 1914, xiii, 1-16. Ueber die Behandlung des Stotterns. Zentralbl. f. Psychoanal., Wiesb., 1912-1913, iii, 469-500. Ueber die Klangverhältnisse in der Nase beim Sprechen und Singen. Arch. f. Laryngol. u. Rhin., Berl., 1911, xxv, 420-434. Zur Diagnose des Simulierten Stotterns. Wien. Med. Wchnschr., 1911, lxi, 2812-2820. Zur Differentialdiagnose Zwischen Frischem Traumatischen und Veraltetem Stottern. Med. Klin., Berl., 1916, xii, 694-696. Zur Frage der Entstehung des Tonischen Stotterns und zur Frage der Wirkungslosigkeit des Elektrischen Stromes in den Meisten Fällen von Stottern. Med. Klin., Berl., 1917, xiii, 448. Zur Frage des Wesens der Stotterbewegungen. Med. Klin., Berl., 1916, xii, 1028. Zur Pathologie des Stotterns. Arch. f. Exper. u. Klin.

Phonet., Berl., 1913, i, 372-380. Zur Psychologie des Simulierten Stotterns. Wien. Med. Wchnschr., 1911, lxi, 2884.

GARBINI, G.: Disartria e Nucleo Lenticolare. Ann. di. Manic. Prov. de Perugia, 1914, vii, 3-29.

GODFREY, C. G.: Stammering from War Shock, Treated by Psychotherapy, Cases. M. J. Australia, 1918, ii, 262.

GOODMAN, H.: Stammering Cured by Hypnotism. Med. J. S. Africa, Johannesb., 1914-1915, x, 95.

GORDON, R. G.: Stammering As It Occurred in War. Bristol Med. Chir. J., 1919, xxxvi, 143.

GOSSET, H.: Le Bégaiement et la Ré-Éducation Motrice. Rev. de Psychothérap., Par., 1911-12, 207.

GUGGENHEIM, L. K.: Stuttering. Interstate M. J., St. Louis, 1917, xxiv, 224-227.

GUTZMANN, H.: Classification of Disturbances in Speech. Berl. Klin. Wchnschr., 1913, L, 1193. Sprachheilkunde. 2 Aufl., Berl., 1912.

HATFIELD, M. L.: How to Stop Stammering. Chicago, 1919.

HOEPFNER, T.: Psychologisches ueber Stottern und Sprechen. Ztschr. f. Psychoth. u. Med. Psychol., Stuttg., 1911, iii, 264-289. Stottern als. Assoziative Aphasie. Ztschr. f. Pathopsychol., Leipz., 1911-12, i, 448-453. Ueber die Disposition der Stotterer-Psyche zu Asozialer Entwickelung. Arch. f. Krim.-Anthrop. (etc.), Leipz., 1912, xlix, 149-174. Von den Symptomen des Stotterns. Beiträge zur Kenntnis des Stotterns für den Praktischen Arzt. Therap. d. Gegenw., Berl., 1912, liii, 356-360.

HORN, H.: Selected Chapters in the Study of Speech Disturbances. Calif. State J. M., San Fran., 1912, x, 26.

HUTT, C. W.: Census of Stammering Children in Brighton Public Elementary Schools. School Hyg., Lond., 1916, vii, 119-124.

KENYON, E. L.: Case of Injured Palate with Stammering. Ill. M. J., Chicago, 1914, xxvi. 623. Nature and Origin of Stammering. LARYNGOSCOPE, St. Louis, 1919. xxix, 639. Problem of Stammering and Its Solution. Journal-Lancet., Minneap., 1918. xxxviii, 555-561. Also LARYNGOSCOPE, St. Louis, 1918, xxviii, 666-678. Problem of the Stammering Child. Ill. M. J., Chicago, 1914, xxvi. 95-101. Stammerer and Army Service. J. Am. M. Assoc., Chicago, 1917, lxix, 664. Stammering as Disorder of Speech Dependent on Conditions of Child Development. Am. J. Dis. Child., 1919, xviii, 537.

LAUBI, O.: Ueber den Wert der Psychoanalyse für Aetiologie und Therapie des Stotterns und Verwandter Sprachstörungen. Zentralbl. f. Psychoanal., Wiesb., 1913-1914, iv, 41-52.

LEARY, T. G.: Stammering and Corrective Measures. M. J. Australia, 1918, ii, 259-262. Treatment of Stammering. Austral. M. J., 1911, i, 204.

LECLERE, A.: Un cas de Traitement Psychothérapique du Bégaiement. J. de Psychol. Norm. et. Path., Par., 1914, xi, 235-241.

LEVINE, M.: Preliminary Report on the Treatment of Stuttering, Stammering and Lisping in a New York School. Psychol. Clin., Phila., 1912-13, vi, 93-106..

LILLIENTHAL, W.: Cause of Stammering. Med. Times, N. Y., 1911, xxxix, 302. Treatment of Stammering. Med. Times, N. Y., 1911, xxxix, 325.

LINHARES, A.: A Cura de Gagueira. Tribuna Med., Rio de Jan., 1916, xxii, 25; 41.

MCBEATH, H. F.: Correction of Impediments of Speech in Our Public Schools. J. Am. M. Assoc., Chicago, 1913, lxi, 1610-1612.

MCCREADY, E. B.: Mechanism of Speech and Some Speech Defects. Va. Med. Semi-Monthly, Richmond, 1910-11, xv, 321-323.

MACEY, M. S.: Two Cases of Hysteric Stuttering in Children. Med. Rev. of Rev., 1914, xx, 251-253.

McHATTIE, T. J. T.: Educational Treatment of Stammering Children. School Hyg., Lond., 1911, ii, 308-314. Discussion, 322-326.

MACMAHON, C.: Paper on the Curative Treatment of Stammering. School Hyg., Lond., 1911, ii, 315-321. Shell Shock and Other Affections of Voice and Speech. Practitioner, 1917, xcviii, 427.

MAKUEN, G. H.: Defects of Speech. LARYNGOSCOPE, St. Louis, 1913, xxiii, 234-236. Demonstration of Defects of Speech. Penn. M. J., Athens, 1913-14, xvii, 275-277.

MAKUEN, G. H.: Psychology of Stammering. J. Nerv. and Ment. Dis., N. Y., 1916, xliii, 68-72. Report of Three Unusual Cases of Stammering. Ann. Otol., Rhin. and Laryngol., St. Louis, 1913, xxii, 776-781. Also Tr. Am. Laryngol., Rhinol. and Otol. Soc., New Bedford, Mass., 1913 xix, 304-309. Study of 1000 Cases of Stammering with Special Reference to the Etiology and Treatment of the Affection. Therap. Gaz. (etc.), Detroit, 1914, n.s., 385-390. Also Tr. Coll. Phys., Phila., 1914, xxxvi, 102-112.

MARAGE: Étude et Traitement du Bégaiement par la Photographie. Compt. Rend. Acad. d. Sc., Par., 1914, clviii, 730-732.

MARTIN, F.: Stammering. Quart. J. Speech Educ., Menasha, Wis., 1919, v, 287-293. Underlying Causes and Method of Correction of Stammering. Med. Rec., N. Y., 1920, xcvii, 914.

MAYER, O.: Zur Kasuistik des Hysterischen Stotterns. J. f. Psychol. u. Neurol., Leipz., 1914, xxi, 15-17.

MEANS, C. S.: Speech Defectives. Ohio S. J. M., 1913, ix, 180.

MEIGE, H.: Les Dysphasies Fonctionnelles; Comment Étudier les Bégaiements. Rev. Neurol., Par., 1913, xxi, pt. ii, 653-668.

MICHAUX et BOON: Deux cas de Bégaiement. Poloclin., Brux., 1913, xxii, 121; 135.

NEUSTAEDTER, M.: Stuttering and Its Treatment. Pediatrics, N. Y.,

MONES, L.: Treating the Stammerer. English J., 1919, viii, 16-27.

NADOLECZNY, M.: Sprach-und Stimmörungen in Kindesalter. Liepz., 1912.

NETKATSCHEFF: Clinic and Psychotherapy of Stammering. Mosk., 1913. 1913, xxv, 285-290.

OSWALD, M. V. O.: Stammering and Voice Defects. Volta Rev., Wash., xxi, 1919, 708-711.

PACINI, A. J. P.: Stammering. Med. Rec., N. Y., 1918, xciii, 800-806.

POOCK VAN BAGEN, N. J.; Stuttering, Its Origin and Treatment. Med. Rec., N. Y., 1911, lxxx, 464-466.

PRIDEAUX, P.: Stammering in War Psychoneuroses. Lancet, 1919, i, 217,

PRIESTLEY, J.: Common Speech Disorders of Childhood. Brit. J. Child. Dis., Lond., 1916, xiii, 104-113.

PULIDO: La Emocion en Patologia Verbal, y Particularmente en la Tartamudez. An. R. Acad. de Med., Madrid, 1913, xxxiii, 310-330.

REED, R.: Clinical Study of Stammering. Lancet-Clinic, Cinn., 1915, cxiv, 352-361.

ROBBINS, S. D.: Plethysmographic Study of Shock and Stammering. Am. J. Physiol., Balt., 1919, xlviii, 285-330. Plethsymographic Study of Shock and Stammering in a Trephined Stammerer, Am. J. Physiol., Balt., 1920, lii, 168.

ROSENTHAL, F.: Ueber die Behandlung des Stotterns. Wien. Klin. Wchnschr., 1917, xxx, 1123. Zur Theorie und Praxis der Behandlung des Stotterns. Wien. Klin. Wchnschr., 1918, xxxi, 725-731.

ROTHE, K. C.: Die Stoische Philosophie als Mittel Psychicher Beeinflussung Stotterer. Ztschr. f. d. ges. Neur. u. Psychiat., Berl., 1917, orig., xxxvi, 54-58. Ueber Verlegenheits-Sprachstörungen und ihre Beziehungen

zum Ausgebildeten Stottern und Poltern. Zentralbl. f. Psychoanal., Wiesb., 1912-13, iii, 519-524.

SATTNER, B.: Eine Psychoanalytische Studie an Einem Stotterer. Zentralbl. f. Psychoanal., Wiesb., 1911-12, ii, 18-26.

SCRIPTURE, E. W.: Care of Speech Defectives. Med. Rec., N. Y., 1913, lxxxiii, 339. Nature of Stuttering. Lancet, 1916, ii, 494. Speech in Disease. Volta Rev., Wash., 1917, xix, 314. Stuttering and Lisping. N. Y., 1912. Treatment of Stuttering. J. Am. M. Assoc., Chicago, 1911, lvi, 1168-1171.

SCRIPTURE, M. K.: Diagnosis of Speech Defects. LARYNGOSCOPE, St. Louis, 1918, xxviii, 78-88. Etiology of Speech Defects. LARYNGOSCOPE, St. Louis, 1918, xxviii, 12-46. Manual of Exercises for the Correction of Speech Disorders. Phila., 1919. Stuttering and Stammering. LARYNGOSCOPE, St. Louis, 1918, xxviii, 156-174. Therapy of Speech Defects. LARYNGOSCOPE, St. Louis, 1918, xxviii, 817-848.

SCRIPTURE, M. K., and GLOGAN, O. Speech Conflict . . As An Etiological Factor in Stuttering. . . J. Nerv. and Ment. Dis., Lancaster, Pa., 1916, xliii, 139-149.

SCRIPTURE, M. K., and JACKSON, E.: Rational Treatment of Stuttering. LARYNGOSCOPE, 1917, xxvii, 74-80.

SIDIS, B.: Psychopathic Aphonia and Catalepsy. J. Abnorm. Psychol., 1917, xii, 100.

SKILLMAN, G. R.: Stammering; Its Cause and Cure. Balt., 1919.

SMITH, E. M.: Amelioration of Stammering and Other Defects of Speech; Special Teaching for Public Elementary School Children. Med. Officer, Lond., 1915, xiv, 217-220.

SOLOMON, M.: Nature and Cause of Stammering. N. Y. M. J., 1920, cxi, 435. Remarks upon Dr. Coriat's paper, "Stammering as a Psychoneurosis." J. Abnorm. Psychol., Bost., 1915, x, 120-137.

SPYKER, S.: Speech Defects, Modern Treatment by Vocal and Articulatory Exercises. N. Orl. M. and S. J., 1911-12, lxiv, 42-47.

STEDMAN, A. B.: Outline of the Elements and Treatment of Stammering. Bost. M. and S. J., 1916, clxxv, 818.

STEIN, L.: Beitrag zur. Methodik der Stottertherapie. Ztschr. f. d. ges. Neurol. u. Psychiat., Berl., 1917, Orig., xxxvi, 451-466.

STEKEL, W.: Ein Fall von Schreib-Stottern. Zentralbl. f. Psychoanal., Wiesb., 1910-11, i, 328-330. Nervöse Angstzustände und ihre Behandlung. 2 Aufl., Berl. u. Wien., 1912.

STELLMAN, L. J.: Mrs. Mabel Farrington Gifford Has Cured Thousands of San Francisco Children of Defective Speech. Sunset, 1919, xlii, 48.

STERTZ: Hysterisches Stottern bei Einem Unfallkranken. Deutsche Med. Wchnschr., Leipz. u. Berl., 1911, xxxvii, 478.

STEVENS, M.: Why Class Work is of Limited Value in the Treatment of Stuttering. Pedagog. Sem., 1917, xxiv, 36-52.

STIVERS, C. G.: Stammering; Its Cause and Treatment. South Calif. Pract., Los Angeles, 1919, xxxiv, 25-27.

STOELTZER, W.: Anteponierende Alliteration als Physiologische Form der Kinderlichen Stammelns. Jahrb. f. Kinderh., Berl., 1911, lxxiv, 192-194.

SUNE Y MEDAN: Presentacio d'un cas de Tartamudesa. Ann de. Med. Butll. mens de l'Acad., Catalunya, Barcel., 1911, v. 340-344.

SWIFT, W. B.: Can Stuttering Be Outgrown? Quart. J. Speech Educ., Menasha, Wis., 1919, v. 368-374. Developmental Psychology of Stuttering. J. Abnorm. Psychol., Bost., 1916, xi, 258-264. Psychologic Analysis of Stuttering. J. Abnorm. Psychol., Bost., 1915, x. 225-235.

SWIFT, W. B. and HEDRICK, J.: Note on Mental Peculiarities as Symptoms in Stuttering. J. Applied Psychol., Worchester, Mass., 1918, ii, 89-91.

SWIFT, W. B. and HERRICK, J.: Sidetracking of Stuttering by "Starters." J. Applied Psychol., Worchester, Mass., 1917, i, 71-83.

TERMAN, L. M.: Does Your Child Stutter? Harp. Week., 1912, lvi, 12.

TITIUS: Ein Beitrag zur Kasuistik des Stotterns. Allg. Ztschr. f. Psychiat. (etc.), Berl., 1914, lxxi, 207-212.

TOMPKINS, E.: Definite Solution of the Stammering Problem. LARYNGOSCOPE, St. Louis, 1919, xxix, 409-419. Knowledge of Stammering for Teachers. J. Educ., 1916, lxxxiv, 524. Left-handedness and Stammering. Quart. J. Speech Educ., Menasha, Wis., 1919, v, 6-11. Perception of Stammering. Quart. J. Speech Educ., Menasha, Wis., 1918, iv, 2090-2095. Problem of the Stammerer. N. Y. M. J., 1917, cvi, 241. Prohibit Stammering. Kindergarten Mag., 1916, xxix, 22. Revolution in the Field of Stammering. English J., 1919, viii, 527-534. School, Society and Stammering. School and Society, 1916, iv, 331-332. Should Hypnotism be Used to Correct Stammering? Med. Rec., N. Y., 1919, xcv, 731-733. Stammerer. Survey, 1918, xl., 377. Stammering. Med. Rec., N. Y., 1918, xciv, 62. Stammering. N. Y. M. J., 1920, cxi 900. Stammering and Consistency. Educa. 1916, xxxvi, 386-388. Stammering and Its Correction in the Home. J. Home Econ., 1916, viii, 606-607. Stammering and Its Extirpation. Pedagog. Sem., 1916, xxiii, 153-174. Stammering and Modern Medicine. Mod. Med., 1919, i, 448. Stammering and the Evidence of Its Inheritance. Sci. Am. Supp., 1918, lxxxv, 186-187. Stammering and the Ladies. Sci. Am. Supp., 1918, lxxxvi, 10. Stammering As It Is. Med. Rec., N. Y., 1920, xcviii, 935. Stammering Discussions. J. Abnorm. Psychol., Bost., 1917, xii, 260-264. Stammering in Connection with Military Service. Am. J. Pub. Health, 1917, vii, 630. Stammering Is Not Amnesia. N. Y. M. J., 1919, cx, 717. Stammering, Its Own Explanation. Sci. Am. Supp., 1916, lxxxi, 96. Stammering Problem Solved. J. Abnorm. Psychol., Bost., 1919, xiv, 349. Stammering Scientifically Considered. Sci. Am. Mo., 1920, ii, 111-113. Treatment of Stammering. Bost. M. and S. J., 1917, clxxvi, 651. Also Med. Rec., N. Y., 1917, xci, 865. Useful Knowledge of Stammering. Am. Med., Burlington, Vt., 1917, n.s., xii 179. When Does Stammering Become Pathological? J. Ophth. and Oto-Laryngol., Chicago, 1917, xi, 321-325.

TOWN, C. H.: Training of a Case of Infantile Stammer. Psychol. Clin., Phila., 1910-11, iv, 136-140.

TRÖMNER, E.: Das Stottern eine Zwangsneurose. Med Klin., Berl., 1914, x, 407-412. Zur Pathogenese des Stotterns. Deutsche Ztschr. f. Nervenh., Leipz., 1913, i, 214-217.

TUCKER, A. C.: Impediments of Speech in Children; How to Overcome Them. Educa., 1914, xxxiv, 354-360.

VENABLES, J. F.: Stammering. Seale Hayne Neurol. Studies, 1920, i, 331.

VON LENZ, J. R.: New Cure for Stuttering. Illus. World, 1917, xxvii, 25-26.

WALLIN, J. E. W.: Theories of Stuttering. J. Applied Psychol., Worchester, Mass., 1917, i, 349-367.

WILE, I. S.: Economic Value of Speech Correction. Med. Rec., N. Y., 1917, xc, 583-585.

TWO NEW TONSIL CRYPT EVACUATORS.

Dr. Henry S. Wieder, Philadelphia.

At the present day, with the great abundance of tonsil instruments that are on the market, one hesitates to bring forth anything new, but the writer has felt for some time that too much attention has been paid to the removal of the tonsils and too little to the diagnosis of their diseased conditions.

The customary examination of the tonsils consists in inserting a tongue depressor in the mouth and seeing if the tonsils are large or small, or rather if they project beyond the pillars of the fauces. Many large tonsils are completely concealed behind the anterior pillar and many tonsils that project almost to the median line are not particularly large, but are displaced toward the midline because there is little or no fossa between the anterior and posterior pillars of the fauces.

However, in the light of modern investigation and the stress that is being placed more and more upon focal infection, it is readily seen that the mere size or hypertrophy of the tonsils is not the most important fact to be determined. It is more essential to determine if the tonsil is a possible focus of infection, the seat of localized purulent accumulations or contains deep crypts having within them deposits of bacteria, desquamated epithelium and food debris which take the form of the characteristic cheesy masses. These are often extruded automatically into the patient's mouth, giving rise to a bitter taste and foul breath.

Entirely too little effort has been put forth in this direction, the only serious attempt that has been made to study the diseases of the tonsils has been by French by means of his tonsilloscope, but unfortunately the training necessary for the successful application of the instrument has deterred others from following in his footsteps. Davis has devised a tonsil evacuator which works on the principle of a comedone extractor, but it is subject to two serious objections. In the opinion of the writer its use is not satisfactory in those cases of long slit-like crypts at the upper pole or for emptying the supratonsillar fossa. Even in those large flat adherent tonsils which do not readily lend themselves to expression from in front, the instrument is too small and sharp and too likely to cause trauma.

It is with these objections in view that the writer has modified the Davis instrument for use in the large flat tonsils by increasing the size of the orifice so that the lumen of the new instrument is of the same size as the outside dimensions of the Davis evacuator. The opening slopes toward the center so as to feed the tonsil into the instrument gradually without injuring the tissues. This constitutes one end of the instrument. The more important and useful end consists of a small crutch arrangement, hollowed on the end and concavo-convex on the flat, so that the end of the instrument corresponds exactly with the curve of the back of the tonsil. By making pressure against the anterior pillar, with the convex surface outward, the tonsil is readily displaced from its bed and practically everted so

that the mouths of the crypts open up and empty their contents. The instrument can then be slid around to the upper pole and more cheesy material expressed. It is surprising to observe the amount of material that can thus be forced out. Purulent pockets can also readily be detected in this manner.

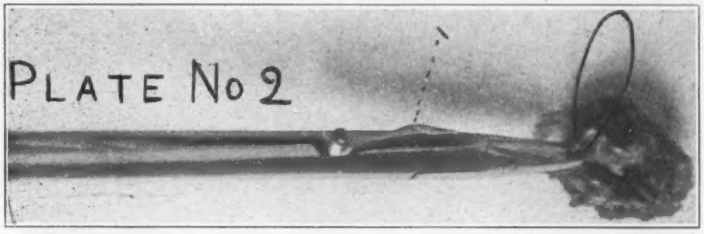
The presence of cheesy deposits or purulent pockets is a far more certain indication for tonsillectomy than mere increased size. This instrument is useful, not only from a diagnostic standpoint, but can be used therapeutically in those individuals in whom tonsillectomy is indicated by these findings, but contra-indicated because of some general systemic condition. Frequently patients can be kept comfortable by periodic emptying of tonsils without their removal.

2035 Chestnut St.

HEMOSTATIC FORCEPS FOR LIGATING VESSELS IN TONSIL FOSSA.

Dr. J. F. Callahan, Brockton, Mass.

The haemostatic forceps illustrated was devised for tying vessels in cavities where operations are being performed and are beyond the reach of the fingers, which are needed in placing the ligature over the end of a hemostat. It is generally conceded that the tonsil fossa is one of the most difficult places to successfully ligate a bleeding point, as well as a region in which more operations are performed than in any other part of the body. One of the chief difficulties in ligating a vessel in the tonsil fossa with the ordinary hemostat is that we must get a very small bite or we will be unable to complete the tie. With hemostat here illustrated, we can take



as large a bite as we wish, which, in my experience, is easier to obtain than a small bite.

Another point in favor of the being able to tie when we have obtained a good bite is that our technique is improved, inasmuch as the hemostat has less chance of becoming detached during the procedure. Instrument as shown in Plate I is threaded by passing ligature under clip (1), which is lifted away from instrument at this point when the instrument is closed. Instrument is then opened and the clip (1) closing, holds the ligature firmly. Ligature is then passed through grooves near end of jaws (2) and then passes down the opposite side of instrument, to be secured beneath clip on handle of instrument. Instrument is held open in a fixed position by a spring between the handles and a restraining pivot in lock.

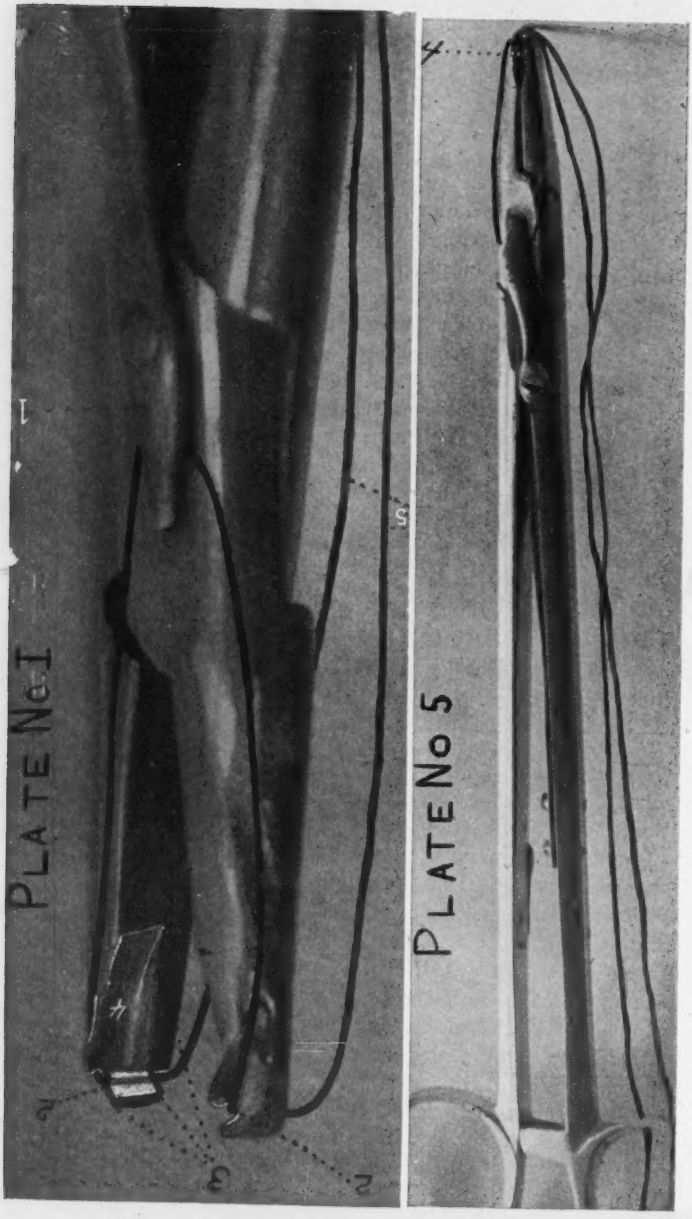


PLATE No I
PLATE No 5

Plate II shows instrument in position grasping bleeding points; ligature then being automatically released from clip (1, Plate I and II). Ligature is now pulled against tissue and into depression in blade at 4 (Plate I and V). This depression at 4 (Plate I and V) is the same depth on both blades. While tissue is firmly held at three points (3, Plate I), the loose tissue in the depression (4, Plate I) allows us to complete the tie while the instrument is still closed. Depression is seen at 4, Plate V, where instrument is closed and extends slightly more than half way across each blade.

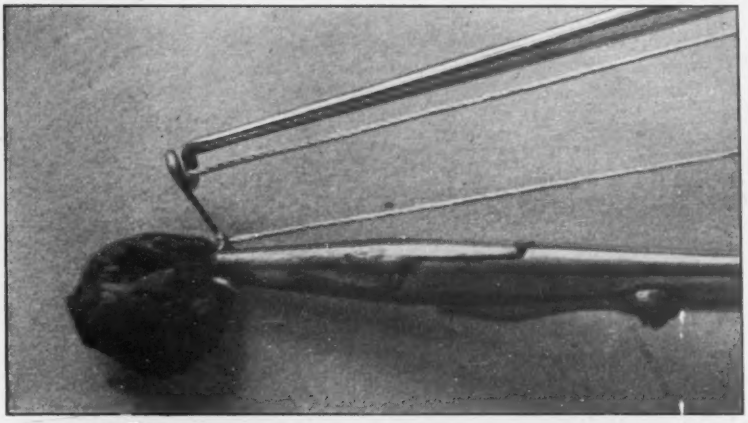


Plate 3.

Plate III shows the ligature carrier securing the knot. The knot may be tied with instrument closed. While we are sure to make good the tie if we have an assistant to remove the instrument when we are completing the tie, there are very few failures recorded to complete the tie while the instrument is closed when No. 12 twisted silk or No. 1 pyoktanin catgut (slightly moistened) were used.

The bite need not extend over two millimeters into jaws in order for it to be past the grooves (2, Plate 1, through which the ligatures pass) and reach that part of the jaws which firmly secures it and makes possible a successful tie.

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SECTION ON OTOLOGY.

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(Continued from p. 658.)

That brings up the question of why cannot that influence on these fibres, the vertical or the horizontal, be due to something different from their anatomical distribution. Dr. Fisher's views on these paths may or may not be correct, but his argument in support of them does not seem to be complete in view of the facts referred to.

In the investigation of these cases, one thing seems to have been decided: those who do pay attention to this mechanism cannot follow Barany altogether in his localization of the different parts of the cerebellum. From the clinical point of view we do not always find that the evidence that we get from the testing of the individual enables us to locate the lesion in the superior or the inferior part of the lateral lobe, or even in the vermis;—that is, in locating the lesion in the cerebellum we have not always succeeded, but have been surprised to find that our deductions were not correct, following the data we have.

Dr Strauss said that today he would be rather loath to make a diagnosis from the tests as to exactly what portion of the lateral lobe was affected,—though he did not know whether or not Dr Fisher had come to that conclusion.

Regarding vertical nystagmus, he said that the neurologists consider it as evidence of stem lesion. Vertical nystagmus is seen in so many diseases which are organic, and which are known positively to be due to organic lesions in the stem, that when such a symptom is noted on testing it is felt that it is probably always indicative of a lesion of the brain stem.

Dr. Strauss agreed with Dr. Fisher that it was a crime to diagnose a case of ponto cerebellar tumor as one of multiple sclerosis, for if there was one point in neurology where a definite diagnosis can be made it is with an angle tumor. It has been found, however, that tumors in the cerebellum give symptoms which to neurologists cannot be differentiated from those of an angle tumor, particularly when it is a large tumor and presses on the eighth nerve as well as in the angle.

In a recently observed case there was considerable discussion as to whether it was a tumor of the pons or of the angle. There was a loss of caloric response in the canal, with deafness; there was also evidence of involvement in the pons, especially since there was anesthesia of the fifth nerve of a segmental type,—that means the track of the trigeminal nerve in the brain-stem and not the main nerve which is affected. The question came up: Did she have a ponto-facial angle, or only an angle tumor? She did not have choked disc. Subsequently she developed a choked disc, and was sent to Cushing, and he made a diagnosis of tumor in the middle fossa, because he had found tumors growing from the tempero-sphenoidal lobe and pushing back to the angle subtentorially. His examination, however, revealed nothing,—but at a subsequent operation he exposed the ponto-facial angle and found a tumor there.

This is a field of medicine which possesses extreme importance. It belongs partly to the otologists, but is just as important to the neurologists; in fact, it seems probable that the neurologists can get more from it than can the otologists.

DR. K. WINFIELD NEY opened his remarks by saying, that the value of those differential tests concerning vestibular function would be increased with greater clinical and pathological experience; that they would be of greater assistance in differential diagnosis when the evidence presented tonight has been fully substantiated by pathological proofs. Their greatest value at the present time consists in the elimination of cerebellar lesions and in the examination of the peripheral vestibular mechanism. There are certain things which I believe must be considered in reference to the determination of changes of an inhibitory nature in the various tracts, and I believe that it is possible to have partial lesion of the peripheral portion of the eighth nerve which might be very confusing in drawing conclusions regarding the inhibition of impulses through various tracts. Our experience with peripheral nerves in other parts of the body have been such as to impress us with the importance of a partial interruption of certain fibers in a nerve trunk. In the average mixed motor sensory nerve it is possible to find a great variation in the reaction of its various funicular constituents; *i. e.*, compression of such a nerve may be sufficient to inhibit impulses through those fibers which subserve the function of sensation, while those fibers governing certain muscles will be entirely uninvolved. It depends entirely upon the way the pressure affects the nerve, and they vary greatly, from time to time. This phenomenon must be considered in the interpretation of findings where the eighth nerve is involved, and it is quite possible as a result of injury or pressure to find a physiological inhibition of isolated tracts, depending entirely upon which portion of the nerve is subjected to the greatest amount of pressure. In such a condition we might find an involvement of the eighth nerve where only the auditory portion may be involved, or only a portion of the vestibular fibers, which I believe would lead to great confusion and might give one the impression of a cerebral lesion with involvement of an individual tract. I think this could probably only be differentiated by a study of repeated consecutive findings. The change from time to time, such as an increase in the amount of pressure might cause a marked variation in symptoms and greatly influence the value of these tests from a standpoint of localization.

In tumors of the eighth nerve where the involvement is usually the result of changes developing in some portion of the nerve sheath, it may be readily understood how it would be possible to get an involvement of certain of the funicular constituents, without the involvement of others. This is certainly true in early lesions, and I think it is of great importance that sufficient recognition be given to the elimination of partial lesions of the peripheral portion of the eighth nerve before concluding that the individual tracts of the vestibular mechanism are involved in the brain.

I would like to ask Dr. Fisher if he has any way at the present time of differentiating, in the presence of a partial lesion, between a peripheral and central involvement?

DR. CHARLES A. MCKENDREE said that he had greatly enjoyed Dr. Fisher's communication with reference to the vestibular mechanisms and felt that in his present communication, as well as in his splendid previous contributions Dr. Fisher has added greatly to the differential diagnosis and interpretation of central lesions. Otology is certainly to be congratulated upon the magnificent results it has obtained, especially during the past ten years with reference to the correct understanding of vestibular control. During this period neurology has been making considerable progress regarding localization, particularly as the result of the work done by Mills, Weisenberg and Tilney.

It is probably difficult for otologists to realize how rarely purulent lesions are seen in the wards of general hospitals to which neurologists have access. Abscess of the brain and cerebellum and septic meningitis due to otitic origin are quite infrequent in the neurologist's clinical experience. Dr. McKendree felt very strongly that there was need for more team-work between the otologist and the neurologist, especially in circum-

scribed purulent lesions. By such co-operation the neurologist might derive great assistance in his attempt to correlate important clinical data with reference to localization. Whenever the diagnosis is concerned with cerebral lesions involving the vestibular or cochlear pathways, the neurologist cannot get on without the otologist, and when the otologist once determines that there is central disturbance, he should enlist the services of a competent neurologist.

In furthering the study of localization, neurologists would be very much benefited if they had the privilege of seeing such conditions more frequently. In reviewing the literature dealing with purulent lesions of the brain and cerebellum as described by otologists, Dr. McKendree had been somewhat disappointed to find in the majority of cases that when there had been great difficulty in making differential diagnosis, the finer technique of neurological interpretation had been quite ignored. Many of these interesting problems must have given rise to certain valuable neurological signs of which there was no mention in the accounts.

He suggested that probably in most cases where the vestibular determinations indicate a central lesion, thorough examination by a competent neurologist ought to determine the existence of such a lesion, and lead to accurate localization, with the aid of the otologist's interpretation.

He referred to the well known manifestations of asynergia major and minor and hoped that in the future such determinations would be made and recorded for the purpose of correlating, to better advantage, clinical data of cerebellar lesions. It would be of particular advantage to the neurologist to know the exact location of such lesions, together with pathological studies when available, with the hope that the functional significance of the various lobes of the cerebellum might definitely be brought to light.

Dr. Phillips said it was a pleasure to listen to clear-cut statements such as made by Dr. Fisher, and it was gratifying to the otologists to see the atmosphere clearing up somewhat in facing this type of cases. There has been a very marked improvement in the ideas concerning these conditions and in the results obtained with them during the last few years. As Dr. McKendree had said, those who work in the large otological centres get their material from such a different source that they see the purulent types more frequently than the tumor abscess types, though many of the latter cases also are seen, and the diagnosis is often made along the lines shown by Dr. Fisher. It would be well if the younger otologists would do more work along this line and help the development of the science of neurology.

Dr. Friesner said that, despite his long friendship with Dr. Fisher, his knowledge of and familiarity with the latter's valuable work, he could not bring himself to agree entirely with what had been said, due largely, no doubt, to a difference of experiences. He did not feel that there has been sufficient advance in the study of pathological material to substantiate the statements made in regard to the separate pathways in the stem. Some years ago he had taken the view which he still holds,—that the changes in the nerve itself inflammatory perhaps, or due to increased intracranial pressure, may for some reason cause a difference in the permiability of the two sets of nerves. For instance, he has seen a case of lead poisoning in which the vertical canals of one side did not react, though there was a perfectly normal response from the other side. Dr. Strauss had mentioned a case of specific neuritis, and Dr. Fisher himself had mentioned a case of specific neuritis, in which one only external canal reaction was not normal,—so that there is evidence that toxic substances exercise a selective action on parts of each vestibular nerve bundle. Now and again we have seen, with increased intra-cranial pressure, losses of reaction from the vertical canal. Why they should not act under these conditions is not clear. Philogenetically, it should be the external canals whose function should be abolished, for the vertical canals are developed earliest.

Dr. Friesner said that speaking from the otological point of view he wished to refer to the aid obtained from the neurologist. Time and again when the otologists are confronted with the problem of intra-cranial localization in acute inflammatory lesions, the neurologist does not give any help. That is not his fault, any more than the lack of ability to make the diagnosis is the fault of the otologist. The conditions differ decidedly from those that obtain in slow-growing non-inflammatory tumors,—even to the extent that the evidence of pyramidal tract lesion may be crossed; despite a right-sided temporal abscess there may be symptoms referable to the opposite, *i. e.*, left side of the cerebrum. Localization in the acute intracranial suppurative lesions must be a very difficult thing,—judging not only from the failures of the otologists, but also from the fact that so many excellent neurologists when consulted in these cases have been unable to help.

Dr. Alfred Braun wished to emphasize the point brought out by Dr. Friesner, namely, that in the localization of acute suppurative processes of the brain, the otologist gets very little help from the neurologist. He cited the case of a girl upon whom he had done a simple mastoid operation last winter. She did well for five weeks and then suddenly developed a severe headache, paresis of the sixth nerve on the side of the mastoid and an attack of Jacksonian epilepsy of the opposite side. A very able neurologist saw her and made a diagnosis of cerebral abscess. The following day her symptoms improved so much that it was decided not to do anything. A week later there was marked dysmetria of the arm on the side of the mastoid. A diagnosis of cerebellar abscess was made, and a large abscess was found in the lateral lobe of the cerebellum.

Dr. Mark J. Gottlieb asked why Dr. Fisher placed the caption falling in the section of the chart which had to do with douching. He had observed on testing vestibular mechanism in some cases of chronic purulent otitis media that very often these patients would exhibit absence of past-pointing, particularly with that arm which is on the side of the lesion. These patients do not complain of headache, the hearing is good and the eye grounds normal. It would be interesting to know to what Dr. Fisher thinks this condition is due. In a case recently tested, the patient had a chronic purulent otitis media in the right ear with a large polyp completely occluding the external auditory canal; in this case she did not past-point to the left with her right hand and the vertical canals of the other ear did not respond to stimulation by either turning or douching. The horizontal canals gave normal responses. This patient had no headache, eye grounds were normal and auditory nerves on both sides functionating. Such findings are common and I am sure Dr. Fisher has seen them often.

Dr. Sautter said he had been doing this work for the past five or six years in Dr. Dench's service of the Neurological Institute, where something like three to five hundred cases are examined annually, and although he had been very enthusiastic upon the subject, he had not been able to find any help in differentiating these two pathways that Dr. Fisher had spoken of. So far as the tests are concerned, however, the elimination of the eighth nerve is very important when making them. In a case seen recently the patient presented all the symptoms of sinus thrombosis, with a temperature of 106° and a history of a discharging ear since childhood, being now nineteen years of age. He was tested very carefully, because of a slow pulse, ranging between 50 and 65, with the possibility of a cerebellar abscess. In examining the left vertical canals, he overpointed in opposite direction 10 inches and there was no overpointing when stimulating horizontal. He was tested twice on the same day, in order to determine whether these facts were correct, and each time the symptoms were the same,—the over-pointing was perverted ten or twelve inches to the right on stimulation of the vertical canal, and no over-pointing on the horizontal. A jugular resection was done on the patient, and his temperature has gone down; but he may have some cerebellar complication now, for his pulse is still low.

Dr. Sautter expressed the hope that Dr. Fisher would speak a little more fully in regard to perverted past pointing. He had also stated that nausea and vomiting were more frequently observed in cerebral than in cerebellar conditions.

Dr. Danziger said that in his service at the Montefiore Home and Hospital he had a veritable gold mine of interesting cases of the central nervous system,—quite a number of which he tested with the calorimeters. While these examinations should be made with a perfect mechanical exactness, it becomes evident at once that even with crude methods valuable information can be obtained. Subjecting a number of blind patients to the caloric tests will show immediately that the quick component of the nystagmus is not dependent on vision. On testing a woman with a left sided subcortical tumor of the brain, the caloric test produced a deviation of the eye to the left without any quick component of the nystagmus. A woman who had a paralysis of the right external rectus muscle of the eye, when subjected to the cold water test, showed a typical vestibular nystagmus,—showing that the nerve pathway of the vestibular apparatus was untouched and that the lesion must be looked for somewhere between the cortex and the medulla.

Dr. Fisher, in closing the discussion, said that the Philadelphia neurologists had all been very helpful and had worked hand in hand with him. Dr. Mills, Dr. Weisenburg and Dr. Spiller had given their time and attention freely,—thus making this work possible. It had always been a wonder to him that he who was no neurologist could once in a while diagnose these conditions correctly. There certainly must be something in the method if a blind man can score a hit with it.

Much had been said in the discussion about the differentiation of the pathways from the two sets of canals. No one who had made a great many of these examinations could have failed being impressed with the cases where in a perfectly normal ear the vertical canals give no response, while the horizontal canal does. The otologist must prove first of all that the vertical canals themselves are normal. He had douched cases where the vertical canals gave no nystagmus, no vertigo or past pointing, but the patients were made seasick, which proved that the canals were functioning. How are such phenomena to be explained?

Dr. Strauss had brought out the point that certain lesions do have a selective action for certain fibres, and Dr. Friesner had called attention to the fact that certain toxins have a similar selective action. Granting that certain diseases have a predilection for certain types of fibres, it is not so clear that it could be a satisfactory explanation for the phenomenon under discussion. Let us recall for a moment the following: there are two types of fibres originating from the vertical semicircular canals—one type for eye-movements and another type for motion-sensing (those producing vertigo). The firbes from the horizontal semicircular canals are also of this dual type. If the contention of Dr. Strauss, that there is no neuraxial differentiation of all these fibres be correct, then we ought to expect the lesion to single out one *type* of fibre—the same type of fibre in both sets of canals—we would then have either the eye-movement fibres singled out for suppression or the motion-sensing fibres, but always from both sets of canals. Observations, however, show nothing of this sort. Both types of fibres from a set of canals are suppressed in their entirety, which leads us to believe that it is rather a question of location than susceptibility because of certain structure.

Dr. Fisher said that at best this question was one of purely academic interest and that some day research along histologic lines will definitely determine this question. The fact is that we do observe this phenomenon clinically and that this has served so far as a very useful clinical formula in the neuro-otologic study of cases.

As to Dr. Strauss' question of how accurate cerebellar localization can be made with this method, Dr. Fisher said that no attempt is usually made for very accurate focal localization. Occasionally the findings might

suggest an involvement of either the right or the left side of the cerebellum, depending on whether it is the right or the left side of the body that shows an interference with correct past-pointing after stimulation. But the more accurate localization must naturally be left to the neurologist.

Replying to the question of the significance of the failure of one arm to past-point after douching, Dr. Fisher said that no one should attempt to suggest a lesion from one sign only. One must have an entire "phenomenon-complex" before any such suggestion can be made. He could not say definitely, just what was the significance of the failure of one arm to past-point properly, but he did know that to assume the existence of a lesion on this evidence alone would be very unwise and dangerous.

Replying to the question as to when a case was otologic or neurologic, Dr. Fisher said that the minute an otologist suspects that a case with vestibular symptoms he is studying, is one of central lesion, it should be promptly referred to a neurologist. Under no circumstances ought the otologist continue with it, since his means of examination are limited to the ear only, whereas the neurologist has many other types of examination available which would enable him to treat the case properly.

Dr. Fisher said that he hoped to see the time when every suspected brain case would have an examination of its vestibular mechanism. While it is true that some few cases are made somewhat uncomfortable by an ear examination—that they will occasionally become nauseated and will even vomit, still the value of the information derived from such an examination far outweighs any such inconsiderable drawbacks. Neurologists and intracranial surgeons do not hesitate to perform lumbar punctures in the hope of obtaining some useful information, although a puncture of the spine is a more or less formidable procedure. Compared with this, an ear examination is a trifle and in the vast majority of cases would probably throw more light on the case.

Replying to the question asked by Dr. Gotlieb, as to why the "falling" test is found on the chart in the space reserved for "douching" and not in that for "turning," Dr. Fisher said that with the patient sitting in a turning chair with the head in the usual position, the turning stimulates the horizontal canals in a horizontal plane and the vertigo induced is in the same horizontal plane. Naturally there can be no falling in a horizontal plane. Falling can only be elicited when the induced vertigo is in an up-and-down plane. The douching test, with a patient sitting in an upright position, stimulates the vertical canals in just such an up-and-down plane and the falling response can be very conveniently observed.

SECTION ON RHINOLOGY AND LARYNGOLOGY.

February 23, 1921

Singers Showing Deformity Following Tonsillectomy. Dr. L. D. Alexander.

Dr. L. D. Alexander, Jr. presented three patients whose throats showed marked deformity following tonsillectomy, in spite of which condition there was no apparent impairment of the singing voice. It was his opinion that the question of tonsillectomy in singers should be governed by the same judgment as in any other patient, as he had never seen any noticeable impairment of the singing voice due to properly performed tonsil operation.

DISCUSSION.

Dr. Hays said he could not let these three cases pass without comment. Most of the men had for a long while been under the impression that it is not fair to operate on the tonsils of singers, but he, like Dr. Alexander, felt that the indications for operation on singers were very definite in those patients that are really suffering from some generalized disease for which the tonsils are responsible. The general health comes first,

and the question of operation must be considered. He then cited the case of a singer upon whom he had operated two years ago, an extremely nervous woman upon whom he did an excellent tonsillectomy, and who had improved mentally as well as physically, but with no impairment of the singing voice. It took her a full year with the greatest persuasion before she would utter a single singing note. Another patient refused operation and later had one of his tonsils removed abroad. Later he came to Dr. Hays and insisted on having the other tonsil removed, and his singing voice is excellent. Much depends upon the impression made upon the mind of the singer as to whether or not the voice is going to be impaired.

DR. JONES said he had had his patients run a scale imediately after the operation, and do that three or four times an hour until they recovered their confidence, and he had had very good results, there being fewer adhesions of pillars by doing this.

DR. HURD said that he had never seen the removal of tonsils from a singer's throat which had injured the voice in the least. If anything, the voice had been improved in resonance, volume and range. The indications for removal are the same as in non-singers, but he feels that it should be properly done with no injury to the muscles about the capsule or loss of the mucous membrane. Also, he has the singer absolutely refrain from singing until the throat is entirely healed, and then start slowly to train the stylo glossus, the stylo pharyngeus and constrictor to function in their new and normal positions, so that there will not be false action of these accessory muscles established that night lead to faulty method.

DR. HAMON SMITH said that he had always been cautious in advising the removal of tonsils in singers whose voice and method were permanently established, for fear of altering same.

In all singers, however, upon whom he had operated for the removal of tonsils the voice had not only increased in volume, but in register.

In cases of diseased tonsils constantly secreting and this secretion dropping into the larynx it becomes difficult, on the part of the individual, to remove it from the larynx and the effort of doing so thickens the cords. He could see no reason why singers should not be treated the same as any one else, particularly in the course of their instruction. When the singers, however, had reached the age of forty years or more and had permanently established their methods, unless there were grave constitutional symptoms warranting it, he believed that treatment was the best course.

DR. J. H. ABRAHAM said that he had operated upon quite a number of public singers and that he agreed with what had been said—that tonsils should be operated upon when the pathological conditions warrant it. Among the last public singers he had operated upon was the brother of R and Miss V. The latter has now a perfect singing voice and is the representative of the Edison Company, giving concerts. Previous to her operation she was subject to repeated attacks of rhino-pharyngo-laryngitis, and also had beginning singer's nodules. She had very large tonsils, the crypts of which were filled with cholesteatomatous masses. Since her operation, about a year and a half ago, she has been free from colds. No one could ask for a better result. It is not so much the operation on the singer but the form of the operation selected, and the technic more than anything else. One is dealing with a patient who requires the use of the musculature of the pharynx more than others. Recently he had operated on a singer from out West, and at the same time upon two other adults, all three of whom had been previously operated upon during the last few years. The singer had had an operation which he does not practice. The pathological lesions were as follows: On the left side the base of the tonsil remained and the left posterior pillar was completely adherent to the pharynx, not only by scar tissue but welded up into a mass of lymphoid tissue so that the patient could not use her voice. It required a very delicate operation to remove the

adhesions and to remove the base from the posterior pillar, for it was attached all the way down. After this dissection the pillar was liberated and the soft palate instead of being drawn to the left side by the adhesion was restored to a normal position. Her voice is now practically perfect.

DR. IMPERATORI said that he had been looking over the literature of the subject, and learned that some sixty or seventy years ago a physician made a practice of removing the uvula. He did this deliberately and many persons went to his office in the hotel here in the city. The writer reported that the treatment did not seem to do very much harm, neither did it do any good, but still the patients flocked to him. These patients shown by Dr. Alexander evidently had not suffered much harm; still we do not start out today with the intention of doing a uvulectomy.

Bi-Lateral Abductor Paralysis of the Larynx. Dr. E. L. Pratt.

(History sent to LARYNGOSCOPE by Dr. Lederman a few days after meeting.)

A Case of Bilateral Abductro Paralysis.* Dr. E. L. Pratt.

*Read at the Laryngological Section, New York Academy of Medicine, February 23, 1921.

J. H., 35 years. Admitted December 9, 1920.

C. C. Difficulty in breathing, hoarseness, cough.

P. I. Onset. suddenly one week ago.

Has had difficulty in sleeping because of smothered feeling.

Personal History. Married; has wife and three children all living and well. No history of TBC. or malignancy in the family. Is a plumber by trade; has not been handling or using lead in any form lately. No recent illnesses of note except influenza three years ago. Alcohol, drugs and venereal history denied.

Physical Examination.

Larynx: Shows a bilateral abductor paralysis.

Heart and Lungs: (Dr. Norris). There is a loud ringing aortic second sound over the base which is also heard over a considerable area. This is very suggestive of aortitis eith dilatation. No pulsation seen or felt, no tug, no pulse inequality. Soft blowing systolic murmur over aortic area. Lungs show emphysema and there is considerable retro-mandbrial dullness with bronchial breath sounds over manubrium. Knee jerks brisk and equal. Pupils react sluggishly to light, better to accommodation. The third, sixth and seventh nerves normal. No ataxia, no afferent sensory disturbances. Nothing found on physical examination to explain the lesion.

Neurological Examination: Pupils sluggish to light left more than right; both respond better to accomodation. Other cranial nerves negative except for a slight ptosis of the left upper lid which patient claims has always been present. Upper extremeties show lively deep reflexes, no sensory changes, no ataxia; abdominal and cremasteric reflexes present. Lower extremeties also show lively reflexes with a tendency to an ankle clonus. No Babinski. No evidence of paresis.

Radiograph of Chest: No infiltration or consolidation in either lung, no pleural effusion, no tuberculosis. There are pleural adhesions at the base of the right lung. Heart shadow normal. Diagnosis: thickened pleura right lung.

Blood and Spinal Wassermann negative.

Spinal fluid: Clear, average pressure, Globulin negative, Noguchi negative.

Sputum: Negative for TBC.; urine negative.

Summary. A suggestion of an Aotitis with dilation (Dr. Norris). Thickened pleura right lung (X-ray). Pupils which react sluggishly to light.

This patient was first seen at the Vanderbilt Clinic on December 9, 1920. He came to the clinic to seek the cause of, and relief from, recurring attacks of what he termed "smothering sensations." These

attacks were especially frequent on lying down at night, and also caused him discomfort during the day when he attempted any unusual exertion.

The first unusual sign noticed was his peculiar speech. He was only slightly hoarse, but he spoke in a jerky manner, uttering a few words at a time with a short interval between them during which he would stop to take a deep inspiration. He spoke in a monotone with scarcely any modulation to his voice.

Examination of his larynx by the indirect method showed a bilateral abductor paralysis. The cords were very nearly approximated in the midline with only a small chink between them. There was no movement of either cord on respiration but on phonation there was a slight yet appreciable abduction of both cords.

Because of the severity of his attacks of dyspnea he was referred to Dr. Coakley's service at Bellevue Hospital for investigation and observation. He remained in the hospital about ten days without any appreciable change in his physical signs, but with considerable improvement in his symptoms. His dyspnea became much less while he remained quiet and at the end of three days in the hospital he was able to sleep all night with no discomfort or dyspnea.

Since leaving the hospital I have seen him once or twice a week at the clinic and have found no change in his laryngeal condition. He has had a few mild attacks of dyspnea but no severe ones and has been able to work steadily as a janitor since leaving the hospital.

I am presenting this case mainly for the discussion of two features, (1) the etiology and (2) the treatment.

According to Semon-Rosenbach the abductor fibres of the recurrent laryngeal nerve are the first to become affected in a paralysis of this nerve, the adductor fibres being not only less vulnerable but also showing a greater recuperative power. Reisen, Russell and others have demonstrated that the abductor and adductor fibres of the recurrent laryngeal run in separate bundles throughout the whole course of the nerve and occupy separate centres in the medulla on the floor of the fourth ventricle.

Since the publication of these papers the law of Semon-Rosenbach has been subjected to many experimental and clinical tests—most notably by Dufourmental in his thesis for the French Academy, published in 1914. With very few exceptions this law appears to be accepted as true. Numerous cases have been reported of complete paralysis terminating in an abductor paralysis alone—notably Semon's case where, after almost complete loss of voice caused by a complete paralysis, the patient was able to resume his occupation as a professional singer.

In attempting to explain the etiology of the case presented here we must consider, as in all such cases, whether the lesion might be a cortical or bulbar one, a peripheral neuritis, a myopathic condition or a mechanical fixation of the crico-arytenoid articulation.

1. A cortical lesion causing a bilateral abductor paralysis has never been demonstrated. To produce it would require a bilateral identical lesion involving the cortical centres or the motor fibres passing from them to the medulla. The destruction of one cortical centre produces no result—the opposite centre continues to act. Moreover, it has been proved that a unilateral irritation of one cortical centre causes a bilateral movement but of adduction only—never abduction. That is, it is the purposive movement of phonation; abduction, or the more automatic movement of respiration is represented mainly in the medulla.

2. If the lesion is a bulbar one it must also be a bilateral one situated on either side of the floor of the fourth ventricle and confined only to the nerve endings of the abductor fibres.

3. In explanation of a peripheral lesion in the mediastinum we have only the doubtful suspicion of an aortitis (not demonstrated by radiograph) and a thickened pleura on the right (so diagnosed by the radiographer). It would, however, be a most peculiar coincidence to have two such separate and distinct lesions manifest themselves at exactly the

same time each causing only sufficient pressure to involve the abductor fibres.

Tracheal compression by tumor, etc. (Direct examination.)

4. There is nothing in the history to suggest a myopathic or toxic cause—no history of a rheumatic diathesis and nothing to lead one to suspect any of the mineral poisons such as lead, or the vegetable ones such as alcohol, belladonna or cocain.

5. Fixation of the crico-arytenoid articulation on both sides is not likely in this case as there is distinct adduction in both cords and the point of fixation of the cords in abduction is symmetrical; neither is there swelling or tenderness about the joint.

6. That the exact etiology of these cases is often impossible to determine even at autopsy is well known. Of 150 cases examined post mortem by Avellis the cause was demonstrable in only 35-56 per cent.

Most writers agree that the prognosis in most bilateral abductor paralysis is grave, that the sudden onset of severe dyspnea followed by asphyxia is not at all uncommon. What therefore should be the treatment recommended to cases like the present one? This patient has refused any surgical intervention and I cannot help feeling that he is in a constantly threatened condition. The wearing of a tracheotomy tube would remove this threat and by keeping it plugged up during the day his voice would be about as good as at present. At night or during exertion of any kind which induces dyspnea the plug can be removed.

DISCUSSION.

DR. DOUGHERTY said he could not add anything to the etiology of these cases, but he could add two chapters to the mystery. In his hospital practice, a couple of years ago, a young woman had been referred to his service by one of the attending medical men with a history of an absolutely normal chest, no disease of the thoracic viscera whatever that might cause pressure. The neurologist could find no neurological cause for the condition, but she had a decided bilateral paralysis of the abductor muscles. Unfortunately she left the hospital a few weeks later and was lost sight of, so no further report could be made.

Some years previous, he had showed another patient before the Section, a man 35 years of age, giving almost the exact history of Dr. Pratt's case excepting that his chest findings were absolutely normal. The plates showed nothing and the neurologists could find nothing to account for the paralysis. His history and dyspnea was the same as in the case presented tonight. His dyspnea did not persist, and at times he did not feel that he had any trouble in his larynx. He was seen occasionally during the past eight years, and an unsuccessful effort had been made to get in touch with him in connection with this case. His family, however, reported that he was still about the same; that he had drifted from one physician to another, but apparently was in about as good condition as ever. His attacks come on and last for a few days and then pass off.

DR. THURBER told of a case of one-sided paralysis which developed very suddenly in a patient after climbing the leaning tower of Pisa. The etiology in the case proved to be an unrecognized attack of diphtheria which had occurred three months before the patient left this country; the attack was so slight that it was not noted at the time. Her case until that history was obtained was just as blind as this one. She made an uneventful recovery, although the one-sided paralysis was very marked.

In another case, the patient was the captain of a Hudson river steamboat, and had a bilateral abduction paralysis. He claimed that it was due to shouting at his stevedores. He recovered absolutely after two months.

DR. FREUDENTHAL said he felt convinced that most of these cases were tabetic in origin. There are always from three to five cases of this sort at the Montifiori Home, as it might least be expected in any hospital with many tabetic patients. On the other hand, in a case such as demonstrated tonight, it might be found out later that the patient was suf-

fering from tabes or some similar disease. There was only one safe thing to do, to perform a tracheotomy; but he had observed patients with similar conditions who lived from six to ten years with some dyspnea, but they did not exert themselves, as they were blind and unable to walk. If Dr. Pratt's patient would lead a sedentary life, he might go on fairly well for a long time without a tracheotomy.

The Etiology and Treatment of Pulmonary Abscess. H. L. Lynah.

DR. SCHWERDFEGER: It was a great pleasure to hear Dr. Lynah's presentation of these cases of lung abscess, more particularly because it was my privilege to examine a number of them at the Lenox Hill Hospital. One of the causes which he did not mention was empyema, especially of the interlobar variety. The diagnosis of abscess of the lung is usually made from the symptoms. Patients expectorate a large amount of foul-smelling sputum which contains elements of lung destruction. It is particularly characteristic when the expectoration occurs periodically. There is usually clubbing of the fingers. The history is very frequently suggestive, particularly when the symptoms follow an operation on the upper respiratory tract or after a pneumatic process. Other symptoms such as cough, pain, fever, dyspnea, and hemoptysis are not characteristic. The physical signs are usually few. Those most commonly found are dullness with diminution of the breath sounds and voice, and a moderate number of roles. Occasionally bronchial breathing is heard, with increased voice. Definite signs of a cavity are present only when these cavities are superficially located, but this is the exception. The signs may vary, depending upon whether the lung is examined when the cavity is full or when it is empty. The physical signs are so few and indefinite because the abscess cavity is usually surrounded by more or less normal lung tissue. The differential diagnosis from a tuberculous cavity usually offers no difficulty. These cavities form in the midst of an already diseased lung and there are abundant physical signs. Tubercle bacilli can usually be demonstrated, either by examining the sputum or by guinea pig inoculation. Usually where a tuberculous process has advanced to the stage of cavitation, physical signs can be found in the lower lung. The Wassermann reaction should always be made in these cases. A breaking down tumor of the lung must always be kept in mind. The X-ray and the bronchoscope are the means by which the diagnosis is made with more certainty, especially as to the size, number, and location of the cavities.

Abscesses following operations on the nasopharynx are not embolic, because the symptoms do not occur suddenly, because they are not on the periphery where we nearly always find embolic pneumonias, and because there are no other emboli present in other parts of the body. There is no reason why emboli should attack the lung alone. The history of a case of one of the patients which Dr. Lynah showed will give us a better idea of the course which these cases follow than any other means of description.

Boy, 24, admitted to the Lenox Hill Hospital, May 31, 1920, for an arthritis, the acute symptoms of which had subsided but there were still present some joint pains. He gave a history of two attacks of tonsillitis, was ambulant and free from fever. A tonsillectomy was performed on June 5. Discharged on June 12, in good condition. On the day of his discharge he began to cough. Two days later, the cough became productive (nine days after the operation). June 16, he returned with the arthritic pains. The source of infection of this arthritis was now the lung and not the tonsils as before. Re-admitted to the hospital on June 18. Chief complaints: cough and expectoration. The sputum was foul-smelling, and the joints were painful. Examination showed a moderate number of moist rales in the right chest anteriorly—and no other changes —which disappeared in four or five days. The X-ray showed a cavity in the lower part of the upper lobe. On July 1, there was slight dullness anteriorly, extending from the second to the fourth ribs. There were

no rales nor other physical signs, and this dullness was still present on July 31, when he was discharged as cured. The amount of sputum at first was about 100 cc. daily, which gradually increased to 500 cc. daily. After beginning bronchoscopic treatment, this amount lessened and on discharge from the hospital he had no expectoration.

The Treatment: Medically, there is very little treatment. We can teach these patients the position in which they can best empty their cavities. If there are marked symptoms of sepsis, operation should be advised without delay. Occasionally, an individual should be sent to another climate, but only when all the conveniences and medical care can be obtained which is as good or better than that at home. I have had no experience with collapse of the lung by artificial pneumothorax, but would not hesitate to recommend this method of procedure in a case of deep-seated cavity which would be difficult to reach by surgery and where bronchoscopic treatment had been unsuccessful.

Unfortunately tonsillectomies are not regarded by the laity and by many of our colleagues as a serious major operation. Our patients are frequently observed very poorly before operation, sometimes the appointments being made weeks in advance and the patient is not seen again until a few minutes before operation. As a minimum preoperative observation I would demand a twenty-four hour record of the temperature, an examination of the chest and of the urine. In our general hospitals the narcotizers and the assistants are usually not the most experienced men on the staff, these being busy with the general surgeon. A good anesthesia is of greatest importance and it must be emphasized that a satisfactory general anesthetizer may be an unsatisfactory one for tonsil operations. The assistant should be well trained and permanent and not one who only assists at such operations occasionally.

DISCUSSION.

DR. W. H. STEWART: The speaker's first experience in outlining the bronchial tree with bismuth solution was purely accidental. In October, 1915, while fluroscoping an old man with an oesophageal obstruction, he observed some of the bismuth paste passing from the oesophagus directly through a fistulous opening, a portion of the paste passing down into the lower bronchi. A roentgenogram was immediately taken. The patient had three or four coughing spells, bringing up particles of bismuth paste, and the following day he seemed none the worse for his experience. The examination was repeated about a week later, with no ill effects.

Upon investigation the speaker found that a number of similar cases had been reported. It occurred to him at that time that, with proper precautions, the injection of opaque substances into the lung through the bronchoscope, could be safely undertaken.

It was evident that advancements along these lines were slowly being made, for in 1917 Dr. Sidney Yankauer treated a case of bronchictasis by direct application of iodin solution to the diseased area through the bronchoscope. In conjunction with Dr. Willy Meyer and Dr. Yankauer, this case was carefully watched roentgenographically until complete recovery occurred.

Between 1915 and 1920 two cases of tracheo-esophageal fistula came under our observation, in which the main bronchial tree on both sides was outlined by bismuth paste escaping from the esophagus through a fistula into the trachea. In early 1920, the same phenomena occurred in a case of carcinoma of the esophagus, located just above the arch, complicated by laryngeal paralysis; also, in a patient suffering from carcinoma involving the laryngo-pharynx. In both these cases the bismuth paste entered through the trachea beneath the epiglottis, which was imperfectly closed. Repeated roentgen examinations of these two patients did not cause any ill effects.

During the speaker's army experience at Biltmore, N. C., while examining patients suffering from chronic empyema, he frequently injected bismuth solutions into the empyemic cavities which had direct communi-

cation through a pleura-pulmonary fistula with a branch bronchus. The bismuth solution would permeate many of the bronchial branches, be retained long enough to enable him to obtain satisfactory roentgenograms, and then would be expectorated. Such an occurrence, whether accidental or intentional, did not seem to seriously disturb the patient.

The experience mentioned above strengthened his belief that if accidental, inhalation could occur without danger, injections might be done deliberately, and, combined with roentgen examination, be used for diagnostic purposes.

Dr. C. Jackson, of Philadelphia, during 1918, reported a case before the American Laryngological Association, in which the main bronchi on the right side had been outlined roentgenographically, after insufflating dry bismuth through the scope.

Drs. J. C. Bullowa and C. Gottlieb, of New York, in 1919, reported some experimental studies on living animals in which the bronchi had been injected with bismuth and barium mixtures, roentgenographic observations of which brought out details of the bronchial tree heretofore never thought possible. They were able to demonstrate a "wave-like" peristaltic action in the bronchi and trachea

So far as we are able to ascertain, it was not until early in 1920 that any successful efforts were made roentgenographically to outline lung cavities after the injection of opaque substances through the bronchoscope. In May, 1920, Dr. Lynah of New York, presented a short preliminary report on the subject before the American Laryngological Association; he reported two cases of lung abscess which had been successfully mapped out roentgenographically after the injection, bronchoscopically, of aqueous and oily solutions of bismuth-subcarbonate, into the area of diseased lung; both these patients have since been as well as ever, and have been repeatedly examined by the authors without ill effects; the cavities in all were beautifully outlined by this method.

Dr. Richard Jordan said he had been associated with Dr. Lynah in this work for about a year and enjoyed it immensely. Comparatively few men are working in this field, and many have been kept from it by a misunderstanding in regard to the apparent difficulty of the procedure. With the Jackson instruments, however, a gentle hand, and any amount of patience, the procedure is not over difficult, and more of the younger men ought to take it up. It has its reward in itself, and the results are very gratifying.

The time required for the treatment is considerable. Each patient requires half an hour, more or less. Very little cocain or morphin is required. Children do not require any anesthesia whatever, and many adults prefer to be bronchoscoped without it, considering that the after effects of the anesthetic are more unpleasant than the discomfort of the treatment. The introduction of the Jackson tube is difficult only under abnormal conditions. The most disagreeable part of the treatment is the withdrawal of the copious malodorous discharge. It requires considerable time and patience. When that is done, it is usually possible to find the offending bronchus and its branches. These can also be sucked out, and at later sittings injections of bismuth or other preparations can be made. Lately, a new colloid silver solution has been tried out at the Lenox Hill Hospital in infected surgical cases with very remarkable results. I have tried it after radical mastoid operations, especially in children and the prompt results have been surprisingly good.

The same preparation we have injected in these lung abscesses, with apparently good results. On the whole, however, the material on which it has been tried at the hospital during the past months has been too small to prove very much. The two cases, which were completely cured, were injected with bismuth. Both were acute cases and we were fortunate in getting at them some four or five weeks after the incidence of the abscess. Both had had tonsil operations, and both had an abscess in the left upper lobe where the bronchus showed an edematous stricture. After the stricture had been passed by a small calibre bronchoscope the im-

provement was immediate and was very marked, and both patients made excellent recovery. It is possible with the Jackson instruments to use a very small tube; this is a great comfort to the patient and also advantageous to the operator, who can enter the small branches of the discharging bronchus and thus approach more closely the infected area. It would hardly be possible to use a Killian or Bruening's tube as the light, thrown in from without, is insufficient in long narrow tubes. With the Jackson bronchoscope the illumination of the field is the same, no matter how long the tube or how small its calibre.

The treatment is very fascinating, and no one who tries it patiently will ever regret it.

DR. WILLY MEYER said that it was very wise to arrange the discussion with surgery at the end instead of at the beginning, for in studying these cases it is naturally the medical man who first sees them.

It was most gratifying to hear such an excellent diagnostician as Dr. Schwerdfeger emphasize the important fact of the medical man's ability not only to make the diagnosis of lung abscess but to accurately diagnose its site. The regular sequence in establishing this, calls first, for the regular clinical examination, then for the laboratory analysis, after that for the X-ray man's continuation, and last of all for bronchoscopy.

The subject of the differential diagnosis between abscess and bronchiectasis was one that had been repeatedly discussed in the various surgical societies without, so far, any definite conclusion being reached. Dr. Meyer said that personally he believed that in the case shown by Dr. Lynah in the lantern slides—where, following aspiration in the course of an operation for cholecystectomy, a man developed two distinct cavities in the right upper lobe—that patient had lung abscess; whereas, in the case of the boy who coughed up quantities of foul pus, following the aspiration of a piece of timothy grass eight years previously, the patient had clearly a bronchiectasis of the left lower lobe. In other words, cases where there were one or two, or even three large cavities—if rather acute or subacute, he would call lung abscess; where there were many larger and smaller cavities, of chronic character, in one or more lobes, he would call the condition bronchiectasis.

The most careful clinical examination does not always clear up the diagnosis. One may think the rales are heard on the right side, yet they may be transmitted from the left where the trouble really is. To show the exact location, the X-ray is necessary. But radiology, too, often fails to show clearly the presence of bronchiectasis. Then comes bronchoscopy. The bronchoscopist is the greatest friend to the thoracic surgeon, provided he does not hurt or injure the patient. He will say after his examination: "There is the disease. There is a healthy upper right lobe and a healthy lower lobe, but the middle lobe is diseased." That is the most wonderful thing that has been added to our diagnostic resources. Every hospital that claims standardization ought to have a trained bronchoscopist. If one hospital cannot do that alone, a number of hospitals should combine and do it. Bronchoscopy is a necessity now-a-days. More young colleagues should enter this field and become experts with the bronchoscope and aid the surgeon in this difficult work.

Dr. Meyer remarked that although the subject of the discussion was "Etiology and Treatment," he wished to say a few more words on the subject of diagnosis. The injection of the bronchial system with some opaque material had occupied his mind for a long time. At the time of the International Congress of Surgeons in New York, in 1914, he had read a paper on bronchiectasis in which he ventured to propose the injection of the diseased bronchial tree with collargol. He had spoken then of collargol, not of bismuth, for his idea was to inject into the diseased locality a strong antiseptic liquid which at the same time was opaque. With the aid of the radiologist of the Rockefeller Institute, he had made some experiments by injecting the healthy lung of a dog with collargol and then taking X-ray plates—all, of couse, under general an-

esthesia. The solution used—5 per cent—evidently was not strong enough, and the results were not satisfactory. Those experiments had been the forerunner of this work, and now Dr. Lynah and Dr. Stewart have taken it up with bismuth and were able to show these wonderful results. In cases of bronchial fistula, Emil Beck of Chicago had repeatedly injected bismuth paste, and the patient suffered no harm. Indeed, Beck stated that he had cured one case of bronchial fistula with the injection.

Considering the etiology and treatment, Dr. Meyer said that in his opinion the principal cause of abscess formation in the lung—besides acute traumatism as occurred during the war, and other accidents, and in ordinary civil life, after pneumonia, influenza, etc.—the principal cause of lung abscess was aspiration. Dr. Lynah had spoken of embolism. Decidedly, if there was a plebitis with an infected thrombus, the latter, if driven through the heart into the lung, would produce a central acute abscess in one of the lobes. These patients are very often sick after an initial chill, and need an operation. If the lung is found to be not yet attached to the chest wall, it ought to be stitched to it and the abscess then incised immediately or twenty-four hours afterward. Solid foreign bodies are aspirated into the bronchial tree and often cause lung suppuration. Fluids are quickly aspirated into the lung tissue. This occurs during general anesthesia at operations, also during stomach lavage—the patients not being in the right posture in bed.

Aspiration of blood during a tonsillectomy is also a frequent cause of abscess of the lung. The men who are doing tonsillectomies must learn to aspirate the blood from the fauces during the aspiration by suction.

Probably many of the unexplained cases of lung abscess after pneumonia are due to the fact that these patients in their severe sickness aspirated into their lungs matter that should have been expectorated. All this produces lung suppuration.

Dr. Lynah had spoken of lung abscesses in boys who went swimming in our East or North River and aspirated water into their lungs, and the pus showed the colon bacillus in pure cultures. That shows how far behind New York is in her sanitary conditions. This wonderful city of ours still drains all its waste into the two rivers! That ought to be changed. Our young people ought to be able to go into the water without running the risk of lung abscess formation after aspiration of some water into their lungs.

Thoracic surgery is yet in its infancy, but we are learning from day to day. Fortunately we have such men as Dr. Lynah and Dr. Yankauer doing this work here in New York; we have seen that even advanced cases of lung suppuration can be bridged over a critical stage by repeated aspirations, and some can, it seems, thus be cured. These cases have come to be on the border line between medicine and surgery. If aspiration with local treatment does not cure them, they become surgical and must be operated by cutting into the abscess cavity and draining, or by cutting out the diseased portion.

Dr. Meyer stated that he had a series of such patients cured by pneumomectomy. Lobectomy is still a serious operation, with at least fifty per cent of mortality. All must work to make it safe. The hope is that we will learn to do so by getting better control of the pulmonary pedicle with special clamps, dealing with the pedicle of the lung as is done with the kidney in nephrectomy, and will learn not only to control the hemorrhage but to close the bronchial stumps air-tight and thus steer the patient's ship with greater safety through the operation and the first crucial days of the after treatment.

Dr. Lilienthal said that embolic abscess should be operated upon by drainage at the earliest possible moment. No lung abscess should be aspirated from the outside of the chest, because in withdrawing the needle from a septic focus in which malignant anerobes are present, a culture tract through the chest wall is made. He has seen sev-

eral patients die of gangrenous phlegmon of the chest wall following punctures of lung abscess.

The X-ray and endoscopy are better and safer diagnostic methods. Rather than aspirate he would make an exploratory wide thoracotomy.

What has been said of embolic abscesses holds true also of direct or acute unilocular collections of pus within the lung.

The sponge-like abscesses resulting from respiratory aspiration, and especially those following tonsillectomy form another problem. In children and rarely in adults these patients may recover under medical treatment. Oftener they may be improved by aspiration and lavage through the bronchoscope. If the patient's condition is intolerable and stationary or is deteriorating and has gone on for many months with copious foul expectoration, lobectomy is necessary if he is to be cured; always provided that the other lung is healthy. Fifty per cent or more of these patients can be cured by this method. Mortality would probably not be so high in selected cases. Dr. Lilienthal had not refused to operate in these otherwise hopeless conditions when the patient and his family earnestly requested the operation and understood its dangers. In one case he had removed nearly the entire right lung. The man was almost moribund at the time but he and his family begged for the chance given by operation. He made an excellent recovery and is working at his occupation, that of secretary. If one fears to operate in cases which are apparently hopeless without surgery, there is nothing more to be said.

Dr. Lilienthal stated that he thought the two-stage method would do much to minimize the danger of lobectomy. At the first stage adhesions of the healthy part of the lung to the chest wall are caused to form and this renders the anesthetic at the second stage safer and simplifies many problems.

(Dr. Samuel Robinson deserves credit for the two-stage suggestion in this form of surgery.)

SECTION ON OTOLOGY.

March 17, 1921.

Progressive Systemic Deafness as an Endocrine Syndrome. Dr. James G. Callison.

Endocrinology in Otology. Charles E. DeM. Sajous, Philadelphia. (By Invitation.)

Published in full in this issue of THE LARYNGOSCOPE.

DISCUSSION.

DR. CHARLES EDWARD PANOFF: "Dr. Callison and Dr. Sajous have so ably cited their cases, that anyone having had the privilege, as we had this evening of hearing them, would come to the conclusion that endocrinology is a very important subject, and that it has a marked bearing, even in so precise and limited a specialty as otology. They have shown us that an understanding of the principles of endocrinology and their application means a better diagnosis, and what is more important as far as the patient is concerned, a better theraphy in otological cases.

Studying a case along the lines of endocrinology, one must bear in mind the following facts:

1. That inherited tendencies have a marked influence on the endocrines of the individual and therefore on his development.
2. That the endocrines control all metabolic processes.
3. That they play an important part in all the vital functions, and
4. That they are intimately co-related and co-operate in their functions.

The co-operation of the endocrine glands seems to be in a better state than that existing amongst the endocrinologists.

The glands are so co-related that when one gets out of order the others get out of order, that when one fails to perform its proper function or functions, the others will endeavor to take up the work of the slacker. The taking up of this additional work may be the cause of their own derangement. It is on account of the co-partnership of the glands, that we are unable to say that this one, or that one, is of a definite thyroid, pituitary or adrenal type. I believe in the theory of pluriglandular derangements and in pluriglandular (extracts) treatment for these derangements. While it is true that all the signs and symptoms that can be discovered in a given case may point to one of the main glands as the chief offender, still I am certain that when one, who has that fine detective ability, so necessary in the making of a proper endocrine diagnosis, sees the case; that he will find clues pointing to the other glands as accessory agents.

The three main glandular systems, the thyroidal, the pituitary and the adrenal have a marked similarity of function:

They are stimulated by and in turn stimulate the nervous system.

They lower carbohydrate tolerance.

They act and react with the reproductive organs.

They are all concerned in body growth, and

They all take part in the process of immunity.

We all know that there are certain classical diseases that are caused or are supposed to be caused by either a hypo or hyper activity of each main gland. Thus we have Cretinism, Myxoedema, Grave's disease, Infantilism, Froelich's syndrome, Acromegaly, Addison's disease, and that group of cases that have for their main symptom hypertension.

In all these diseases we have ear symptoms, and as the readers of the papers have so fully covered this, there is no need of my repeating it. However, they did forget to mention this fact. That while glandular derangements are responsible for certain ear conditions, that ear conditions on the other hand, might be the cause of glandular derangements. Dr. Callison, some time ago, read a paper along these lines before the section, and while few of the members coincided with his views, it raised enough interest in the subject of endocrinology, to make the program of this evening a possibility.

If all otological cases could be placed in the types of diseases represented by the extremes, the work of the endocrinologists and the otologist would be comparatively easy, but the majority of cases that come under observation, belong to that class which I call the pluriglandular temperament. They present a single sign or symptom, or a number of signs and symptoms that belong either to the hypo or hyper condition of the main glands, or they present combinations of these two conditions, or all kinds of combinations of secretory derangement of all the glands. It is the fine diagnosis of these temperamental disorders that makes the study of endocrinology so puzzlying and at the same time so interesting. Once you are able to make the fine distinctions, which can only be acquired by a thorough knowledge of the symptomatology of the extreme diseases, then and only then will you be in a position to say that endocrinology is an important subject and that it has a marked bearing in all the branches of medicine, including otology, the one that we are interested in this evening.

In closing I wish to thank the officers and members of this society for the opportunity of hearing the papers of Dr. Callison and Dr. Sajous and for the privilege of taking part in the discussion."

Dr. Raphael Lewy is of the opinion that the discussion of a paper depends on one's individual conception of the subject. I consider it a privilege to have listened to the paper of Dr. Sajous which is the result of extensive experimental work and a vast clinical observation. Such clinical and scientific paper cannot be discussed within a few minutes.

My conception of the study of endocrinology, and what knowledge that I have acquired in relationship to it, I attribute to the teachings which I have received from the late Dr. Joseph Fraenkel, who, in my opinion, was not only a scientific physician and clinical observer, but also a master in endocrinology, and if I compare some of these teachings that I have retained from this master, I must admit that some of these statements made by Dr. Sajous and his findings are not compatible with the views of my late teacher.

Otosclerosis, if I consider the morphology of the tissues and their physiologic function indicates to me that there exists a disease which is destructive to the intervening tissues and to the adjacent bony articular surfaces constituting a hyperostosis which results in an ankylosis of the stapedio-vestibular articulation. Whatever the conception of the disease may be as to the primary focus and tissue involvement, whether such disease primarily involves the membranous labyrinth or whether the affection involves the bony labyrinth and destroys the cartilaginous element of the bone, replacing it by bone tissue and therefore, causing an ankylosis, I may state that there seems to be a diversity of the opinion among various otopathologists, as to the primary lesion of the disease.

One school among whom there are such observers as Shiller, Sheiber, Haberman, Katz and Tonby seem to be of the opinion that otosclerosis is a sequence of inflammatory or infectious changes in the middle ear. Such observers as Tonby and Katz concur in such opinion, but consider the disease a possible stapedio-vestibular rheumatoid arthritis.

The other school among whom there are Sieberman, Denker, Potlitzer, Petzoldt, are of the opinion that the disease begins in the bony part of the labyrinth and may involve either the stapes and oval window or the ossicles or the semilunar canals, etc., and that the clinical picture depends upon the involvement of the specific parts. If one attributes this disease to a defective endocrine balance, the question arises first: Is there a similar condition in other parts of the anatomy? In answer, I may state: To me the disease resembles, whether it begins in the membranous portion or in the bony portion, an intra-articular lesion of any other joint which in consequence of the destruction of the intervening tissues between two articulating bones results in an ankylosis.

I may state here that I believe that the disease is dominated by a defective thyroid function most likely of the sub-thyroid type and my hypothesis is based on a conception of the normal function of the thyroid which has to do with development and structure of every tissue in the body whatever its origin may be and which also controls metabolism by its power of neutralization of toxic material and the control of the elimination of waste material.

Conclusion: Considering that this disease would come under the category, if we consider the teaching of the older teachers, of imperfect metabolism and therefore, non-sufficient elimination of waste material causing retension of such waste material in the economy and secondary changes in the joints, and knowing that such deficit is governed by the thyroid gland, I would consider that the disease is sub-thyroidal and would most likely in very many cases react to thyroid treatment if given early in the disease. Secondly, if we consider our more recent conceptions based on laboratory findings, that the disease may be secondary to an infection from a primary focus or an infection primarily at the site of disease, we can also advance the hypothesis that the defective thyroid lowers the local resistance toward infection and causes a nidus for the development of bacteria. This would also classify the disease as a sub-thyroid condition. As endocrine pathology means the disassociated balance in the function of several glands, no condition can be treated monoglandular, and, therefore, in the administration of endocrine therapeutics the individual must be carefully studied as well as the local disease.

DR. BANDLER said that when he accepted the invitation to join in the discussion on the papers of the evening with the otologists he was ac-

tuated by two or three impulses, first, that it would give him an opportunity of expressing to Dr. Sajous his appreciation of what was certain to be a masterly review of the subject; and second, that it would give him an opportunity to again call attention to the importance of observing the by-effects when endocrines are administered for any condition—observing all the results and not focusing attention simply on the object sought by giving the prescription. In one instance a patient was sent to him for treatment who also suffered from a peculiar hissing noise which never left her by day. She had a very decided and progressive relative amenorrhoea. It was thought that the general condition might be at the bottom of it, and in the course of the treatment for that condition, the trouble in the ear was relieved, although that was not the objective sought. About three weeks ago she was given 52 mm. of adrenalin, and when she returned to report she said that two days after she had the hypodermic that peculiar noise in her ears ceased for an entire day. She was given the same thing again, and at the end of the week she said that again the hissing noise had ceased—not the first or the second day, but the third day She received the same treatment several times. The last time she reported she said it was absent for two days, the day afterward and the second day.

Dr. Bandler said that the subject of the evening had an especial interest for him, in that he had hoped and had found confirmation of what he believed to be an important point in medicine, namely, that all the sense perversions etc., etc., etc., are anatomically based on a physical disturbance in the end apparatus itself, or the nerves, or centres that receive the impressions. If that be so, what a tremendous import that has to us. To say no longer psychogenic or psychogenetic for all the abnormalities of the mind was no slight step forward.

Two years ago he had stated his belief that we are, after all, mainly the expression of our endocrines. Since then he has become more and more convinced that not only is that true but that it is our duty to impart that truth to our patients at large, and teach them to understand that sense delusions, etc., etc., are not all psychogenic or psychogenetic; in many cases there is an absolute physical basis for it. And so with perversions of the olfactory sense and of the gustatory sense, of the hearing perception, etc. When this is transferred to all the senses and traced to the endocrines, and treated accordingly we would no longer say that dementia precox, maniac depression, insanity, etc., are psychogenetic or psychogenic. It is the province of the medical men to treat these conditions on the basis of bio-chemistry and the endocrines and not to rest the treatment on the principles of psychoanalysis or the application of the Freudian principles.

Dr. Julius J. Hertz said that Dr. Callison had stated that practically all of his cases belonged to the hypothyroid group. There is, however, a large series of cases, probably half of them, that do not belong to that group. Drs. Beck and Pollock of Chicago, had described the same conditions and their series have shown that the cases are due to a hypoadrenal and hypopituitary condition; therefore, we should conclude that the disease is due to a hypothyroid or to a hypoadrenal and hypopituitary condition.

Dr. Hertz said that in his opinion the heredity that is ascribed to these cases is probably due to a deficient endocrine syndrome inherited from the parents, and as Dr. Sajous had pointed out with reference to the adrenals, we know that these patients are more subject to the infections. It would seem that some focal infection or some toxemia is probably the cause of these conditions occurring in a patient who has a hypoendocrine syndrome. He then asked Dr. Callison whether he had any autopsy work to prove this point, or whether this was just conjecture on his part.

As for Dr. Sajous' paper, Dr. Hertz said he did not know that any one could have put so much in a paper of that length, of what he had found in clinical and experimental work.

In his opinion, the conception of treatment by many men are all wrong. When there is a hypocondition of any organ the idea of many men is to feed the patient too much of that substance in which he thinks the patient is lacking. His own conception of the treatment of such a condition is entirely different. Unless an organ is completely destroyed, small doses should be given, not too often repeated, especially with the thyroid, for the drug is not given to replace the absence of a substance but to stimulate the organ and bring it up to its normal function. Kendal and Plummer of the Mayo's Clinic have shown that if you give thyrotoxin, which is the active principle of the thyroid, ten days are required for it to reach its maximum efficiency. For that reason, they give small doses not often repeated, in order to stimulate the organ and bring it up to its maximum activity.

DR. HAYS said that he felt it was necessary for him to speak mainly from the standpoint of the otologist who admits that he knows about as much as the endocrinologists do about this subject because they are constantly disagreeing. The opinions held by most of the endocrinologists makes it difficult to interpret their work for otological purposes. No one in cases of progressive deafness, fails to realize that the body system plays a great part. We are going through certain phases of medicine today, certain crises, it might be said, which are liable to lead us astray. For example, many cases of tinnitus aurium are sent to the psychoanalyist. The history is gone through with from the age of puberty or before, and the patient is given advice which does very little good. We then come to the treatment with radium—radium applications to the Eustachian tube for the cure of tinnitus and deafness, etc.; and finally we come to the question of the endocrines, and we find from these remarks tonight, that not only must we treat the majority of our cases of progressive deafness by endocrines but that the application of the X-rays externally will be sufficient to stimulate the endocrines so that they will do their work on the ear.

Dr. Hays said it seemed to him that in discussing the factors of progressive deafness the otologist takes into account many factors which are of equal importance. There are many conditions, from acute suppurative or catarrhal conditions on through the list, which can be relieved and have been relieved by the methods now employed; and if we are led to use adrenalin for the cure of cases of progressive deafness we are getting far away. When we were using adrenalin for the cure of all kinds of asthma, it was found that adrenalin had injurious effects. Are we now going to employ adrenalin for the cure of tinnitus of supposed endocrinological origin, which might cause some injurious effects? Moreover the use of adrenalin has injurious effects in certain conditions which do not seem to have any relation to endocrinology whatever. It is not right for an otologist to be too critical about using anything for the cure of an otological condition, but we should still stay on the sane path by realizing that endocrinology is worthy of study but that we should still treat our patients in the most commonsense way that we can.

DISCUSSION.

CHARLES F. STOKES, (New York). Based on the belief that the hypophysis is the mastergland of the endocrine system and that it and sometimes other glands are structurally or functionally disordered by the toxins of infectious disease, focal infections, psychic stresses and the like, often leading to developmental abnormalities and other pathological manifestations, an attempt was made to so electronize the pituitary and other glands as to re-establish electronic balance in them thus aiming to bring about a rejuvenation of structure and function which would reach the projicient apparatus of special sense organs and possibly the various channels and tissues of association, correlation and integration.

In my research work of recent years with the X-ray, I have assumed that electrical equilibrium, or shall we say electronic equilibrium, in the human cell structure is a natural and normal condition, indicating a

state of health in the structure and that any upsetting of this equilibrium from any cause means an unnatural and abnormal condition, indicating disease or disorder. In my use of the term "Electrical Equilibrium," I assume, in line with the electron theory, that all forms of matter are exhibitions of electrical phenomena. Experiments at Harvard University in a Coolidge blub in regard to measurement of electronic energy have shown that the frequencies of the emitted rays have a wide range of values but that the upper limit of frequencies is always proportional to the energy of the electron—in short the frequency of the X-ray wave is determined by the energy handed over by moving electrons. The target plays here, the part of a transformer but appears to play no part in the X-ray wave frequency itself. The swiftly moving electrons in the Cathode rays inpinging on the target may be likened to a stream of water from a hose impinging on a flat surface. The result is a splash and the splash is the X-ray. But the great and recent discovery is that this effect is reciprocal. Whereas the swiftly moving electrons on impact with the target produce X-rays, these X-rays striking any other substance, lose their energy, which however, reappears as the energy of moving electrons. Thus, dependent upon the penetrative power of the X-ray, and dependent upon further research into control of X-ray frequency, the possibility, or even the probability, of the X-ray furnishing a means to restore electrical equilibrium or health in cell structure, is clearly indicated.

Anyone interested in the details of these electrical theories may read with interest the 12th Kelvin lecture delivered by Sir Wm. Bragg before the Society of Engineers in London on January 13, 1921.

Referring to this Kelvin lecture in an editorial, "The Electrician" in its issue on January 28, 1921, states: "These investigations, seemingly only of theoretical interest, which have puzzled scientists of world-wide repute, have produced facts which can be turned to the most practical use. We are at least working in the right direction to obtain the true understanding of the great problems of material structure. The Coolidge tube is only the forerunner of a vast series of useful equipment whose design and manufacture has been made possible by the work of the purest scientists."

The following is the technic employed in electronizing the pituitary—auditory region:—heavy lead foil screen, apperture 2¼ inches, chamois for aluminum radiations; 3 mm. aluminum filter; distance from target 2 feet; 8 milliamperes; time 15 to 20 seconds on each side. This dosage may be used from once to twice a week depending upon the character of the response.

With these precautions this practice is freed of danger, particularly, as it is rarely necessary to prolong treatment further than two months. The usual remedial measures should supplement electronization.

Impaired hearing, and deafness, from chronic otitis media, deaf mutism, epilepsy and various disorders from endocrine imbalance have yielded astonishingly good results. In the field of preventive medicine, particularly during convalescence from infections and other diseases, this procedure should be of decided benefit.

At another time when the cases have been sufficiently studied and tabulated, they will be reported in detail.

Dr. Frank Miller said he wished to speak a few words about this wonderful discussion. The Antithesis of Harmony is Hell; he said he supposed he was the one to raise Hell. "The subject of harmony had been preached by Dr. Sajous, and it was a privilege to hear him. Harmony is Love; we love our old things and have carried them out as long as we could."

He then cited the case of a lady who had recently told him that she was being treated by Dr. C. F. Stokes with X-rays. He said to her: "For God's sake, don't! Look at my hands! That has kept me from operating; for years some one else has had to operate for me. Don't do it!"

Later he thought it over and told her to go on with the treatment, and that he would go with her to see it applied, to which she agreed.

Dr. Bandler had said that he had given five drops of Adrenalin to a patient who suffered from roaring in her ears, and it stopped it for three days. "Well, I took a similar case of terrific roaring to Dr. Stokes and it was cured at once by his application."

Since then Dr. Miller had taken 13 cases to Dr. Stokes, ranging from deaf mutism to oto-sclerosis so-called and most of them had been helped or cured." We have found that many times these wonderful things occur, and today there seems to have been struck a point of treatment upon the pituitary which Dr. Miller believes is going to affect materially this stabilizing governor of the equilibration of brain fluids and whose posterior body probably through some inflammatory development, whether infectious or due to deposition about it of rheumatic deposit, either goads it on to overstimulation or decreases its function, causing the various disturbances, swellings, enlargements of bone in various parts of the body; also causing many such diseases as hay fever in certain seasons of the year; cases of anaphylaxis, with all its misery, etc., for in this gland disability we seem to have a cause. The fundamental principle is that of harmony among the encrines. We cannot use them alone, as a single gland in prescribing them; they always act in unities and their unities must be used in harmony. There are tones of musical quality, as Dr. Callison said of hearing; these high notes often vary in quality of sound, which may seem a blank to the ordinary observer, but to a musician are very definite and quite explainable.

Dr. Sajous spoke of treating with Adrenalin and said that it was the great harmonizer, and so it is; but the great engine, the great stabilizer is the hypophysis, or the pituitary body, and when this is stimulated as Dr. Stokes does by the reduced and altered charge of X-ray, interrupted by accurate timed movements for 20 to 30 seconds, we get a new force which stimulates the pituitary to increase its fluidic control and thus stabilizes all neural connections, even to the peri and endo-lymph of the ear, stabilizing, altering into a new form of eqilibration which positively benefits each and every form of case (such as have been mentioned tonight).

I have done this often by a scheme of dactylization which so acts upon the levator and tensor-palati above and the palato-phyrangeous and superior constrictor below as to cause a better circulation of the blood and lymph fluids of the ear, and the various catarrhal discharges due to the frequent and different invasions of the infected Eustachian tube in a most beneficial way, for instance, buzzing of the ear immediately disappears; so has it with this new X-ray current of Dr. Charles F. Stokes, which he has so logically and clearly explained under the well-known findings of Prof. Bragg.

There can be no harm done from the X-ray under careful development; thus far my objection is, as I have always stated, from my own experience; I feel satisfied that Dr. Stokes has thoroughly overcome this dangerous feature; on the strength of that confidence, I have sent him thirteen cases which vary from intense roaring and buzzing in the ear, deafness occasioned from accident and other causes; to those most difficult to treat and diagnose there has been improvement beyond what I have been able to obtain by means of all the electrical combinations, bougieing, etc., etc.

It appears to even my contrary mind that Dr. Stokes has struck what seems to be a wonderfully safe and constructive method for the relief of the conditions mentioned tonight, as well as other parts of the body over which the pituitary presides. I may be talking of another force from another point of view, hence my statement of the antithesis.

Dr. Callison said there were one or two points brought out in the discussion which he wished to answer. He had been accused of describing thyroid types. That he had not done; he had simply described his patients as he saw them day after day in the clinic. If he described

a thyroid type, it was because a patient of that type had become deaf. He does not believe that one can type these cases. If one gland gets out of order the others will be out of order. Many will be both hypo—and hyper—show an instability.

DR. HERTZ had inquired whether any autopsy work had been done to establish these points. Dr. Callison said he believed that the man who looks into the ears of his patients knows better whether they are infiltrated than if he does an autopsy, and that he can determine the amount of infiltration in a drum—not behind it—more accurately than by doing an autopsy on the patient.

No otologist and no endocrinologist would undertake to cure these patients with adrenalin. There is no excuse for that. It may lessen the tinnitus, but it does not cure the condition. If you are going to cure your patients you have to have better oxidations, there is commonsense in that. He was not refusing to do anything that experience has proved useful, but was simply talking about endocrine treatment. He was sure that Dr. Sajous would agree with him in saying that what thyroid and adrenalin do is to cause better oxidation in the body, and that is the reason for using them. Any drug that will accomplish that object will improve the ear condition, and so long as the oxidation continues better the patient will be better.

DR. SAJOUS expressed his appreciation of the invitation to present this subject before the Section, as he made no claim to know anything about otology, but on general principles there was a great deal to be said. Dr. Lewey had said that he was diametrically opposed to the views of the old school, but he certainly would agree that scientific facts are scientific facts, and that one cannot get behind them. Views other than his own had many points on their side which, when better understood, would all be harmonized provided the teachings of sound experimental and clinical evidence were allowed to prevail. The speaker's own views had never been broken down, even though they were nearly two decades old, because he had adhered strictly to this rule, avoiding all deductions that were not based upon a close analysis of the thousands of correlated facts that even the literature prior to 1903 contained. It was a pity that endocrinology was being obscured by schools or theories in which attempts to explain effects observed were not based upon this golden rule.

As to the by-effects, to which Dr. Bandler had referred, Dr. Sajous could recall two cases, one of them a pituitary tumor and another a case of acromegaly in which he had observed enlargement of the lymphatic glands after giving them hypophysis sicca and ergotin. There was enlargement of the glands and the general effect was similar to mumps. Dr. Bandler's appeal to watch and record these by-effects was very important and should be heeded.

Dr. Hertz had referred to the influence of toxemias. In the functions attributed to the thyroid, and to all the endocrines, we should, in the presence of a toxemia, remember that the endocrines are participants in the immune chemistry of the body, and that the functions of these glands are functionally inadequate, unless the toxemia be sufficiently overwhelming to exceed their defensive efficiency when normal. Since the speaker had urged the participation of the thyroid, particularly in the immune chemistry, it had been confirmed repeatedly on the continent of Europe. Its recognition was of material help in practice. Thus, in a personal case, removal of the tonsils, which caused toxemia, caused a thyroid gland as large as half an orange to recede in two weeks; case after case could be cited in which the removal of a toxemia, of focal origin, whether originating in the tonsils, the teeth, the sinuses, the intestines, etc., was sufficient not only to stop hyperthyroidism threatening life, but also a common goitre. So, when speaking of toxemias in this condition, one should remember that the thyroid not only influences metabolism, but likewise the systemic defensive functions.

(To be continued.)